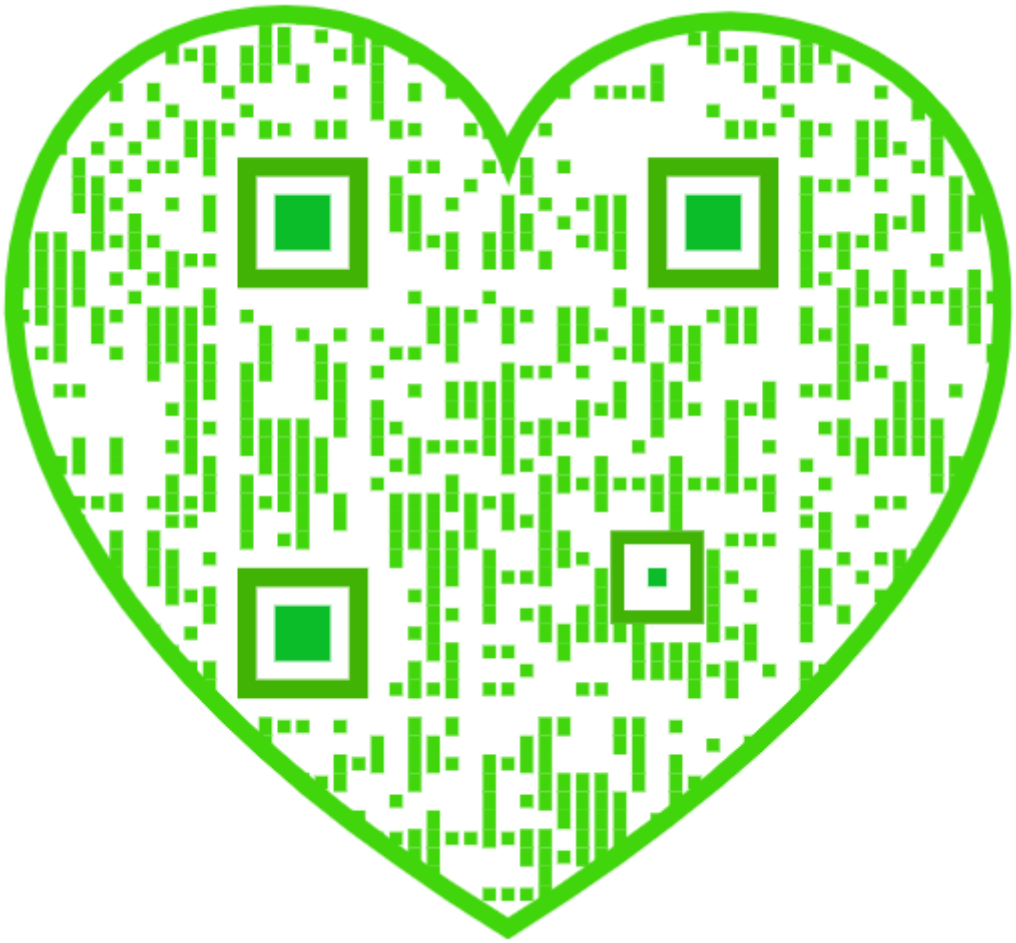

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CLINICAL DIAGNOSIS

BY

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TO HIS ESTEEMED FRIEND

JOSEPH O. HIRSCHFELDER, M. D.,

PROFESSOR OF CLINICAL MEDICINE, COOPER MEDICAL COLLEGE,

THIS HUMBLE VOLUME IS DEDICATED, TO EXPRESS FOR

ACTS OF KINDNESS, THE OBLIGATIONS

AND GRATITUDE OF

—THE AUTHOR.

PREFACE TO THE THIRD EDITION.

THE favorable reception accorded to the previous editions, has induced the author to undertake the revision and enlargement of this work by the addition of many synoptic tables, a chapter on insanity, and a summary of recent methods of diagnosis. The original character of the work has been preserved as an index to or as an abstract of more pretentious works on the subject. "I have gathered a posie of other men's flowers, and nothing but the thread that binds them is mine own."

ALBERT ABRAMS, M.D.

COOPER MEDICAL COLLEGE,
SAN FRANCISCO, Jan. 12, 1894.

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CHAPTER I.

EXAMINATION OF MEDICAL CASES.

In investigating a medical case the following or a similar plan should be pursued, the object being to make a thorough examination, which is facilitated by a definite procedure.

Name. Age. Occupation. Date of Examination.

Patient's history (*anamnesis*). 1. Family history. 2. Diseases of childhood. 3. Previous diseases. 4. Mode of Life. 5. Present Sickness (*date, mode of invasion, etc*).

Objective Examination (*Status praesens*).

General symptoms.

1. Constitution (*bone, muscular and fat development*).
2. Position { in bed—mode of lying; / out of bed—movements.
3. Expression of countenance.
4. Skin (*color, perspiration, œdema, etc*).
5. Temperature.
6. Respiration (*frequency, type, etc*).
7. Pulse (*frequency, rythm, etc*).
8. Tongue.

Then follows the examination of special regions, beginning with the one presumably involved.

Respiratory System.—1. Nose. 2. Larynx. 3. Rythm of respiration. 4. Form of thorax. 5. Type of respiration. 6. Inspection of thorax. 7. Palpation. 8. Percussion. 9. Auscultation. 10. Cough and expectoration.

Circulatory System.—1. Examination of the heart and large arteries (beginning with inspection and following with the other physical signs). 2. Examination of the veins. 3. Examination of the blood.

Digestive System.—1. Lips. 2. Tongue. 3. Mouth. 4. Œsophagus. 5. Stomach. 6. Intestines. 7. Peritoneum. 8. Liver. 9. Spleen. 10. Pancreas. 11. Examination of the stomach contents and fæces.

Genito-Urinary System.—1. Examination of the kidneys, uret- and bladder. 2. Examination of the urine (to be made in all

cases); quantity, specific gravity, reaction, color, odor, sediment; quantity and character by microscopical examination; albumen and sugar.

Nervous System.—1. Examination of the head. 2. Vertebral column. 3. Sensibility. 4. Motility. 5. Examination by means of electricity. 6. Reflexes. 7. Organs of special sense.

Diagnosis. Treatment. Event.

Diathesis. In the examination of patients, the physician often generalizes the symptoms in certain cases, showing that the patient has some peculiar and specific constitutional morbid tendency. The following diatheses are usually referred to:

Phthisical Diathesis.—Clubbing of the finger ends, undue curvature of the nails, red line on the margin of gums, momentary elevation of the skin in percussing the thorax (*myoidema*), flushing of the face (*hectic flush*) paralytic thorax and emaciation.

Apoplectic Diathesis.—Red and congested face, short neck, rigidity of the arteries and obesity.

Gouty Diathesis.—Obesity, varicose veins, oppression in breathing, deformities of the joints with local deposits of uric acid and atheroma of the arteries.

Position of the Body (*decubitus*).—Patients with acute unilateral affections of the chest (*pneumonia, pleuritis, pneumothorax*) usually lie on the affected side to avoid pain during respiration and allow unaffected, to compensate the disturbed functions of affected lung. Patients with pneumonia occasionally lie on the unaffected side in order to avoid pain. The *prone position* may be adopted in certain cases of gastric ulcer. The *head is thrown back* in laryngeal and tracheal disease. In diseases of the heart and lungs when dyspnœa is intense, the *sitting posture* is adopted (*orthopnœa*). *Restlessness* (*jactitation*) occurs during the invasion of acute disease, in delirium and acute mania. In *meningitis* the head is drawn backward. In affections of the *cerebellum* the entire body is often drawn to one side. In circumscribed affections of the *cerebrum* the head may be drawn to one side. If patient is about, the *gait* is an important sign in diagnosis, especially in affections of the nervous system.

Gait in Hemiplegia (paralysis of one lateral half of body).—The arm hangs by affected side and the shoulder droops. At each

step the paralyzed half is lifted to swing weak leg forward. There is a swinging semi-circular movement of paralyzed foot (*sickle-walk*). The shoe is worn off at the outer part of sole and toe.

Gait in Paraplegia (paralysis of lower half of body).—Patient shuffles along without raising either foot from the ground (*hopping gait*).

Gait of Lateral Sclerosis (combination of paresis, muscular rigidity and tremor).—Feet are turned inward and appear glued to the ground. They cross in walking and the knees are liable to become locked together. Weight is thrown first on one cane and then on the other, to lift body so as to move the feet.

Gait of Paralysis Agitans—Tottering and trembling with a tendency to trot. Head bent forward and held stiffly when walking.

Gait of Pseudo-hypertrophic paralysis.—Patients are children. Gait like the *waddling of a duck*. In the erect posture the back is excessively curved (disappears when the patient sits), so that a vertical line dropped from the shoulders falls behind the sacrum. When the patients are placed on the floor great difficulty is experienced in rising, which may however be accomplished by a characteristic movement known as *climbing up the thighs*.

Gait of Tabes Dorsalis.—Legs are flung about in an uncertain way, although the steps are characterized by deliberation. The feet are brought down with the heel projecting (*flopping gait*). Eyes are kept on the ground while walking. Gait due to muscular incoördination.

Gait of Cerebellar Disease.—Likewise due to muscular incoördination. It is staggering like an intoxicated person.

Gait of Cerebro-Spinal Sclerosis.—Unsteady and irregular. No deliberate walking in a straight line, but patient shoots suddenly forward or to one side (*propulsion*).

Gait of Hysterical Hemiplegia.—Leg drawn along sweeping the ground as if lifeless. It is not swung around describing the arc of a circle as in ordinary hemiplegia.

Hemiplegia almost always on the left side, and is developed and passes away suddenly.

Expression of Countenance.—In *Facies Hippocratica, the nose is sharp, eyes hollow, temples collapsed; the ears cold, skin about forehead rough, distended and parched; the color of the whole face being green, black, livid or lead colored.* Described in the words of Hippocrates, this is the physiognomy of approaching death. The same facial expression occurs in *acute collapse*. In *typhoid fever* the expression is dull (*Facies stupida*). In *pneumonia* the face has a dusky flush, in *circulatory disturbances* a bluish

hue. In *cerebral congestion* the face is full and red; in *acute peritonitis* the countenance is pinched. Puffiness of the eyelids may be expressive of *Bright's Disease.*

Examination of the Skin.—1. Changes in color. 2. Perspiration. 3. Œdema. 4. Emphysema. 5. Changes in temperature. *Changes in color*: Pale, red, cyanotic, icteric, bronze skin, gray skin. *Pale color.* Physiological in those not exposed to the air. Temporary in fear and syncope. Occurs *rapidly* in profuse hæmorrhage and in collapse. Often symptomatic of a weakened heart. In diseases of the blood and hæmatopoietic organs: *Chlorosis, pernicious anæmia, leucæmia* and *pseudo-leucæmia.* Also present in malarial cachexia, phthisis, carcinoma and chronic intoxication from mercury and lead. In a number of these conditions not only the pale hue but the color of the skin is characteristic, *e. g.* In *severe anæmia:* waxy lustre and yellowish; *Large white kidney:* light white skin; *Tuberculosis and lead intoxication:* grayish white; *Chlorosis:* greenish; *Diseases of the heart:* dirty yellow; *Cachexia of Carcinoma:* grayish yellow. *Red color* may be caused by: 1. Active dilatation of cutaneous blood vessels. 2. Increase in the quantity of blood. 3. Increase in the blood coloring matter. Universal skin redness occurs in fever, scarlatina and atropine poisoning.

Local redness occurs most frequently in the face. Blushing on one side of the face occurs in the paralytic form of *migraine.* *Hectic flush* in tuberculosis. Anæmia attended with irritability of the heart may render face intensely red.

Cyanosis. Lividity or duskiness of the skin especially marked in the face, lips and finger nails. It occurs when the blood contains too little oxygen and is surcharged with carbonic acid.

Increase of *carbonic acid* in the blood occurs in: 1. Disturbances interfering with the interchange of gases in the lungs; 2. Slowing of the blood current in the capillaries. To the first belong:

a. All conditions leading to a *narrowing of the air passages.* Examples: Obstruction of larynx, compression of trachea and diffuse bronchitis.

b. *Diseases of the lungs* and surroundings. Examples; Emphysema, pleuritic exudation, and abdominal affections interfering with the movements of the diaphragm.

c. *Paralyses, spasms* and diseases of the respiratory muscles. Examples: Bulbar paralysis, tetanus and progressive muscular atrophy. Slowing of the blood current occurs in general *venous stasis*, when from any cause the right ventricle of the heart is weakened, or when the large veins are compressed, just before their entrance into the right auricle (*tumors of mediastinum*). Cyanosis is pathognomonic of *miliary tuberculosis*. Its occurrence in *pneumonia* is an ominous sign. Local cyanosis in the face may be due to extreme cold or occurring in other situations to venous stasis, the result of thrombosis or compression of the veins. The term *morbus cæruleus* is applied to extreme lividity, occurring in congenital malformations of the heart.

In peritonitis, pleuritis and inflammatory affections of the respiratory muscles, the insufficient respirations on account of pain lead to dyspnœa and cyanosis. The character of the former and latter may be correctly diagnosed if they disappear after the administration of narcotics.

Icterus.—A yellow discoloration of the skin occurring over the entire body. The discoloration is first manifest in the *conjunctiva scleræ* and other mucous membranes, and in the skin, where the epidermis is thin.

An object glass, applied with slight pressure to the lips, will render the latter anæmic, and through the glass the yellow color will become more evident. Color varies according to the intensity of the icterus. It is light yellow in mild, and green, or brownish-green in severe forms of the affection (*icterus viridis* and *melasicterus*.) Icterus cannot be detected by gas or lamp light. In examining the conjunctiva the presence of subconjunctival fat must not be mistaken for icterus. Yellow discoloration of the skin may develop after the use of *santonin* or *pieric acid* and its salts. The yellow color of the skin in the beginning of icterus is produced by the biliary pigments in the blood plasma. Later the cells of the *rete malpighii* imbibe the pigment, which fact accounts for the continuance of the icteric coloration after the immediate cause is removed. *The symptoms peculiar to icterus are:* Itching of the skin, minute cutaneous hæmorrhages, slowing of the pulse (due to toxic paresis of the cardiac ganglia), yellow vision (*xanthopsia*) and nervous symptoms (delirium, coma, convulsions, etc.), due to *cholæmia.*

Hepatogenic Icterus (icterus of resorption). Usually results from *bile stasis, i. e.*, any interference with the entrance of bile into the intestines. Causes: *Gastro-duodenal catarrh*, involving *ductus choledochus*, compression of the latter by tumors, or the presence in the same of

ascarides, gall stones, etc.; compression of the hepatic duct, closure of a number of small biliary ducts by intrahepatic gall stones, and, finally, enlargement of the venules of the liver.

The bile is secreted under very low pressure, and even trivial obstructions suffice to prevent its passage into the intestines. After ligating the ductus choledochus in animals, sixty to seventy hours elapse before the conjunctivæ become colored. The diaphragm in contracting subjects the liver to pressure, and is an active factor in forcing the bile from the smaller to the larger biliary ducts. Interference with the movements of the diaphragm (*pleuritis diaphragmatica dextra*) is likely to cause icterus of resorption. The immediate cause of icterus is the accumulation in the blood of biliary pigments.

Hæmatogenic Icterus.—Caused by the destruction of red blood corpuscles. Causes: Acute infectious diseases, particularly pyæmia, yellow fever and pneumonia; after the use of chloroform, ether, chloral, chlorate of potash, etc.

In hæmatogenic icterus the bile freely enters the intestines, so that the stools are not discolored, whereas in *hepatogenic icterus*, the motions being usually free from bile, present a paler appearance than natural (*clayey stool*). In this form of icterus constipation is present, and the fæces are highly offensive. The bile acts normally, both as a purgative and an antiseptic. Examination of the urine is of great importance in differentiating both forms of icterus. (See urine.)

Weil's Disease, also called infectious and epidemic icterus, is an affection characterized by a remittent type of fever, ending by lysis, headache, vertigo and prostration. The pulse is frequent, nausea and vomiting occur in half the cases, and the spleen and liver are enlarged. Icterus is always present. Albuminuria is present in about half the cases. Morbus Weilii may occur sporadically or epidemically.

Icterus Neonatorum occurs at birth and is an example of how sudden diminution of pressure in the portal vein will produce hepatogenic icterus. Normal pressure in the branches of the portal vein is greater than in the neighboring bile ducts; hence a diminution of pressure in the former conduces to bile stasis.

Bronzed Skin (Addison's disease).—Addison, in 1855, called attention to a bronze discoloration of the skin, associated with disease of the supra-renal capsules. This affection is characterized by a gray, brown, or even blackish discoloration of the skin, beginning in parts exposed to the air (face and hands), and then involving parts nor-

mally pigmented, or diffusing itself over the entire body. The nails and conjunctivæ remain unaffected. Circumscribed pigmented spots may develop on the mucous membrane of the lips. The cause of the skin discoloration is a deposit of granular pigment in the cells of the rete malpighii. The constitutional symptoms associated with this disease are: Great feebleness of the muscles and heart, pains in the back and vomiting. It is commonest in young male adults, and is often complicated with phthisis or disease of the vertebræ.

Grayish Discoloration of the Skin (Argyria).—The long continued use of *nitrate of silver* conduces to a deposit of black granules (metallic silver) in the skin. The skin, especially of parts exposed to the light (face and hands), shows the most pronounced discoloration. A similar deposit in the viscera causes the latter to become dark colored. Argyria has been observed to follow even the long continued local application of lunar caustic.

Perspiration.— An increase of the sudoriparous secretion is called *hyperidrosis*, a diminution *hyphidrosis*, and an absence of sweat, *anidrosis*.

Hyperidrosis may be local (*hyperidrosis localis*), confined to one side of the body (*hemidrosis*) or diffused (*hyperidrosis universalis*). Universal hyperidrosis is observed in febrile diseases, tetanus, fever, pain, dyspnœa, collapse, and after the use of diaphoretics and opium. *Acute articular rheumatism* is characterized by profuse diaphoresis. In phthisis (*night sweats*) increased perspiration is frequent. In febrile diseases a fall of temperature is attended with sweating. Local sweating occurs in various neuroses and anatomical lesions of the nervous system. *Anidrosis* is encountered in high continuous fever, and in affections attended by a large loss of water (*diabetes, cholera and contracted kidney*).

Qualitative Change of the Sweat.—Colored sweat (*chromidrosis*) is observed in icterus (yellow sweat). Blue, green and black sweat are said to have been observed. Bloody sweat (*hæmatidrosis*) is really caused by extravasations of blood from the cutaneous blood-vessels. In retarded urinary excretion *urea* may be excreted by the sweat (*uridrosis*), in the form of glistening white scales, which give the reactions of urea.

Œdema of the Skin.—From the capillaries and venules of the skin and subcutaneous tissue a continuous transudation of fluid takes place, which, after subserving

the purposes of nutrition is taken up by the lymphatics and again conveyed to the blood. An abnormal accumulation of this fluid in the substance of the tissues is called *œdema*. When the fluid collects in the greater cavities of the body we have *hydrops* or *dropsy*. It is usual to describe œdema of the integumentary structures as *anasarca*. If the effusion of liquid is general throughout the body, we speak of *general dropsy*, if limited to the peritoneal cavity, it is called *ascites*.

In pronounced œdema other tissues, especially the muscles, contain fluid. Œdematous parts are increased in size, the skin is pale (*pressure of the fluid on the blood-vessels*), smooth, shiny, tense and possessed of a certain transparency. Pressure with the finger on œdematous parts leaves a depression (pathognomonic) called pitting, which soon disappears. When œdema first appears in the feet (*malleoli*), some interference with blood pressure may be assumed, if on the contrary it is first manifested in the eyelids, some constitutional cause (*nephritis*) is probable. Œdema is caused by one of the following conditions: 1. Venous-stasis (*mechanical hydrops*). 2. Altered or watery condition of the blood (*hydræmia*). 3. Inflammation. When œdema is associated with cyanosis and dyspnœa it is usually symptomatic of a non-compensated cardiac lesion. Œdema in venous stasis results from the distended veins, filled to repletion, being unable to take up the fluid normally transuded.

Œdema with Albuminuria.—A combination of these symptoms is called after Richard Bright, *Bright's Disease*. Œdema in albuminuria is explained as follows: The endothelial cells of the small vessels do not normally allow of the passage of any large quantity of plasma. If the nutrition of the endothelium is in any way impoverished as occurs in nephritis owing to the retention in the blood of excretory products, then the blood-vessels become permeable and œdema results.

Œdema with Cachexia.— Œdema without dyspnœa, cyanosis or albuminuria, is usually associated with a vitiated state of the body as occurs in anæmia, phthisis, carcinoma, etc. This œdema is also explained by nutritive disturbances of the blood-vessels.

Œdema with Inflammation.—Usually local and often characteristic of *deep-seated accumulations of pus*. Œdema of one side of the chest is frequently present when the fluid in the pleural cavity is purulent. This œdema is

also called *collateral œdema* and results from increased pressure in the capillaries surrounding the area of inflammation.

Emphysema of the Skin.—This signifies the presence of air in the sub-cutaneous connective tissue. It is characterized by abnormal distension of the skin in certain regions and crepitation is felt and heard on palpation. Pitting is obtained on pressure over the emphysematous parts, but unlike œdema it disappears rapidly. Percussion yields a tympanitic sound.

Two forms of cutaneous emphysema are differentiated, *spontaneous* and *aspirated*. The spontaneous form (rare) is present when gas develops from sub cutaneous extravasations of blood or abscesses. Aspirated emphysema occurs whenever air or gas enters the sub-cutaneous tissue either from without or within. As examples of the former: wounds of the neck or chest. As examples of origin from without: abnormal communications with the sub-cutaneous tissue from any part of the respiratory or alimentary tract.

Temperature of the Skin.—With the hand applied to the skin of a patient, the body temperature can be approximately determined. Palpation of the skin is of value in localizing pathological processes, etc.—See *thermo-palpation.*

CHAPTER II.

TEMPERATURE.

Method of Examination—The temperature of the body may be taken in the axillary space, rectum, vagina or mouth. The thermometer (self-registering) must be very sensitive, compared with a standard one and verified. It should be divided so as to exhibit clearly, fifths of a degree. The vagina or rectum is preferred as representing more nearly the body temperature. When for reasons of delicacy the axillary space is selected, the patient should lie diagonally on the right or left side, his arm firmly compressing the thermometer, which remains in position for at least ten minutes. When the rectum is selected five minutes will suffice. In the rectum, temperature is about 1° F. higher than in the axilla. Before introducing the thermometer into the rectum the bulb is oiled. On the continent of Europe temperature is measured with the scale of *Celsius*, also called *centigrade* (freezing point 0°, boiling, 100°) whereas in England and in the United States the *Fahrenheit* scale is employed (freezing 32°, boiling, 212°).

The scale of centigrade is reduced to that of Fahrenheit by multiplying by 9 and dividing by 5; that of *Reaumur* (Russia and Sweden) to that of Fahrenheit by multiplying by 9 and dividing by 4; and when above zero in either case add 32. Fahrenheit is reduced to either of the preceding by reversing the process. C 100° × 9 = 900 ÷ 5 = 180 + 32 = 212°F.; R. 80° × 9 = 720 ÷ 4 = 180° + 32 = 212°F. The following formula is also employed:

$$N^{\circ}\ C. = \tfrac{4}{5}\ n^{\circ}\ R. = \tfrac{9}{5}\ n^{\circ} + 32^{\circ}\ F.$$

C.	R.	F.
36°	28.5°	96.8°
37°	29.6°	98 6°
38°	30.4°	100.4°
39°	31.2°	102.2°
40°	32.0°	104.0°
41°	32.8°	105.8°

Normal Temperature.—In the axilla this varies between 36.2° C. (97.1° F.) and 37.5° C. (99.5° F.). The temperature is highest *(daily maximum)* in the evening between five and eight, and lowest (*daily minimum*) in

morning between two and six o'clock. The difference between minimum and maximum is about 1° C. (in rare instances 2° C.). Slight temporary elevations of temperature occur after a full meal *(fever of digestion)*, physical exertion and hot baths. A continuous elevation of temperature occurs in fever.

Subnormal Temperature.—This is observed in febrile conditions at the *crisis* and the normal temperature is again attained after one, two or three days. Observed in *collapse* it is accompanied by *diminished cardiac activity*, increased pulse frequency, paleness of the skin and general weakness. It is further observed after hæmorrhages and in the course of chronic, cardiac and pulmonary affections. *Permanent subnormal temperature* (rare) may be encountered in wasting diseases and insane patients.

FEVER.

Fever is not only characterized by a continuous elevation of temperature, but by a symptom complex, the result of increased tissue metamorphosis and functional disturbances of all the organs. Fever may be experimentally produced by the introduction of septic or aseptic matter into the circulation. In infectious diseases the febrile temperature is believed to be caused by the direct action of micro-organisms upon the nerve centres, or by the action of a poison which they develop within the body. It is now believed by many that elevation of temperature attending acute infections is salutary. The growth of the *tubercle bacillus* ceases at a temperature above 41° C. (105.8° F.), and the *spirilla* of relapsing fever disappear at the close of each paroxysm when the temperature reaches 42° C. (107.6° F.). The lesions peculiar to prolonged fever are distributed among the viscera and consist of *granular fatty degenerations* of the cellular elements.

Symptoms of Fever.—Usually the pulse rises synchronously with the temperature, and averages an increase of ten beats for every degree above 98° F.

When the pulse frequency is more than 160 per minute in fever the prognosis is bad. The respirations in fever are increased, there is loss of appetite, increased thirst, digestive disturbances, and diminished secretion of a highly colored urine, with an increase of the solid constituents. The pulse is very *slow* in *meningitis* and *rapid* in *scarlatina;* in uncomplicated *typhoid*, pulse frequency is not usually more than 110. *Herpes* of the lips and nose absent in typhoid and present in *pneumonia* and *meningitis.*

CLASSIFICATION OF TEMPERATURE. (*Wunderlich*).

36° C. (96.8° F.) Temperature of collapse.
37.5° – 38° C. (99.5° – 100 4° F.) Sub-febrile temperature.
38° – 38.5° C. (100.4° – 101.3° F.) Slight fever.
39° C. (102.2° F.) morning; 39.5° C. (103.1° F.) evening. Moderate fever.
39.5° C. (103 1° F.) morning; 40.5° C. (104.9° F.) evening. Considerable fever.
Over 39.5° C. (10 3.1° F.) morning; Over 40.5° C. (104.9° F.) evening. High fever.
Over 41.5° C. (106.7° F.) Hyperpyrexia.

As the temperature rises above 40° C. (104° F.) the gravity of the disease rapidly increases. In certain affections of the nervous system (*tetanus* and *hydrophobia*) the temperature may reach 43°–45° C. (110°–113° F.), and in a few cases this temperature has been exceeded.

Daily Variation of Temperature in Fever.—Analogous to the daily variations in health there is usually in fever an increase of temperature in the evening *(exacerbation)* and a fall in the morning *(remission).* When this is reversed we have the *typus inversus* (usually in phthisis).

Types of Fever.—*Febris continua;* when a daily difference of not more than 1° C. (1.8° F.) exists (temperature usually high). *Febris remittens:* a daily difference of not more than 1.5° C. (2.7° F.).

Febris Intermittens: The fever usually lasts only a few hours *fever paroxysm)* whereas during the rest of the day no fever is present *(apyrexia).*

Febris Recurrens: A continuous fever, lasting from 5 to 7 days, followed by apyrexia from 5 to 8 days; then temperature again rises and lasts from 5 to 7 days, ending by crisis.

Febrile Stages.—*Stadium incrementi:* the stage of rising temperature. *Fastigium:* the stage of highest temperature. *Stadium decrementi:* the stage when the temperature falls.

When the temperature falls quickly we speak of the fever as terminating by *crisis*. When the fall is slow, occupying several days, the termination is by *lysis*. A high rise of temperature preceding crisis is called *perturbatio critica*. Crisis is usually accompanied with profuse perspiration and diminished pulse frequency. *Febris hectica* is a form of the remittent type in which the exacerbations are very high, whereas the remissions go below normal. Hectic fever is observed in *purulent* and *septic conditions*.

Stages of Exanthematous Diseases: 1. *Incubation:* the period from the exposure to the infection, to the outbreak of the disease. 2. *Prodromal stage:* time from the beginning of the fever to the appearance of eruption. 3. *Eruption.* 4. *Defervescence.*

Character of the Fever.—*Febris stupida:* when the patient is apathetic and very quiet. *Febris versatilis:* slight delirium, twitching of the tendons of the wrist *(subsultus tendinum)* and picking at the bed-clothes *(floccitatio)*. These signs are very unfavorable. Fever is further classified as *dynamic* or *sthenic* (full pulse, flushed skin, active delirium) and *adynamic* or *asthenic* (pulse feeble, pale skin, low delirium and great prostration.)

TEMPERATURE AND SYMPTOMS OF THE ACUTE INFECTIOUS DISEASES.

MEASLES—MORBILLI.

Incubation, 10 days. Prodromal stage, 3 days, attended by running at the nose and eyes, sneezing and coughing. Slight fall of temperature on the 2d or 3d day. Eruption (beginning on the face) on the 3d or 4th day, when temperature reaches its highest point. Continuous fever from the 4th to the 7th day. Crisis on the 7th day. Desquamation, 14 days, with very annoying itching. *Eruption* appears first on the face, then spreading to the trunk, and from the trunk to the limbs. It consists of elevated red patches, which tend to assume a circular outline; between these patches the skin is of natural color.

Complications.—Pneumonia, bronchitis and pleuritis.

Sequelæ.—Chronic cough, otorrhœa, enlarged lymphatic glands, etc.

SCARLET FEVER—SCARLATINA.

Incubation, 2 to 4 days. Prodromal stage, 1 to 2 days, commencing with a chill and *rapid* rise of temperature. Eruption on 2d day, with increased temperature. From the 4th day on, temperature falls by *lysis*. *Desquamation* from 4 to 14 days. *Eruption* presents a bright uniform redness, similar to that of a boiled lobster. It appears first on the thorax, abdomen, neck or back. In malignant forms of the disease eruption comes out late, and is either indistinct or dark and livid.

Diagnosis.—Tongue red and papillæ prominent *(strawberry tongue)*, angina, and very rapid pulse.

Complications.—Nephritis with dropsy (usually between the tenth and twentieth day of disease), cerebral symptoms (in children), diphtheria, œdema of glottis, etc.

Sequelæ.—Boils, swelling of lymphatic glands, diarrhœa, otitis, etc.

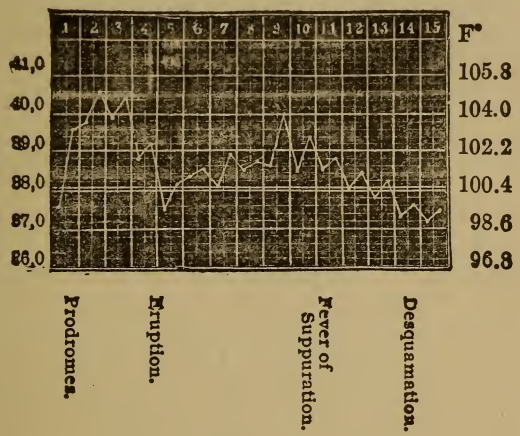
Mortality in epidemics, 50 to 60 per cent.; otherwise about 15 per cent.

SMALL-POX—VARIOLA.

Incubation, 10 to 13 days. Prodromal stage, 2 to 5 days, commencing with a chill and high fever. Eruption on 2d or 3d day, with diminution of fever lasting to the 9th day. From the 9th to the 11th day temperature again rises *(fever of suppuration)*, and is remittent in type. Fever ends by lysis. *Eruption* in most cases appears first on the neck and face, as *red papules*, which feel like shot embedded under the skin. After a day or two the papules becomes *vesicular*, then *purulent*. The pustules may run into one another in grave cases *(confluent small-pox)* or remain isolated *(discrete small-pox)*. A depression in the centre of the pustule

is present *(umbilication)*. Eruption may also affect mouth. In severe forms of the disease hæmorrhages are seen under the skin, as well as inside the pustules *(hæmorrhagic small-pox)*. During invasion severe *lumbar*

Fig. 1. Temperature Chart in Variola



pain is characteristic. The *mortality* in *discrete variola* is about 4 per cent.; in *confluent small-pox* about 50 per cent.

VARIOLOID.

This is modified small-pox, occurring in a person partially protected by vaccination. Usually mild.

Incubation and prodromal stages are the same as in variola, although lighter. Fever of suppuration is absent, *Desquamation* begins on the 9th or 10th day. *Eruption* may resemble that of variola, although it often consists of only a few abortive papules, without vesication or pustulation.

CHICKEN-POX—VARICELLA.

Prodromes usually absent. Fever begins with a chill and lasts until drying of the exanthema (2 to 4 days).

Eruption is vesicular, preceded by red spots. This affection is not prevented by vaccination.

TYPHUS FEVER.

Incubation, 3 to 21 days. Prodromes absent. Begins with a chill and rapid rise of temperature, which is continuous from 13 to 17 days, with slight remissions at the end of the 1st week; and ends by crisis, with *perturbatio critica*. *Eruption* appears from 4th to 7th day and looks like that of measles. The spots *(mulberry rash)* are of a dark tint and very numerous on the trunk and extremities (rare upon the face). The *mortality* is about 25 per cent.

TYPHOID FEVER—TYPHUS ABDOMINALIS.

Incubation, 7 to 21 days. Prodromal stage lasts about a week, and is accompanied by a feeling of *malaise*. In the 1st week temperature rises *slowly*, reaching its highest point in from 4 to 7 days. Then *febris continua* until the 3d week in the mild and the 5th week in the severe forms. Then, while the evening temperature is still high, the morning temperature begins to fall and the fever terminates by lysis, which, in mild cases, is about the 4th week.

Temperature chart in Typhus abdominalis.

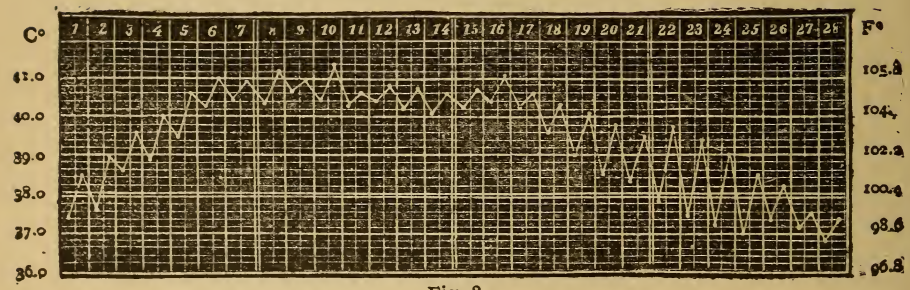


Fig. 2.

Diagnosis.—Tumefaction of spleen, epistaxis, diarrhœa, ileo-cæcal tenderness and gurgling, distension of abdomen *(tympanites)* and nervous disturbances (headache, delir-

ium, somnolence). Eruption (absent in 12 per cent. of cases) appears about the 7th day or later, and consists of small red spots, similar to flea bites. The spots are usually confined to the abdomen and chest, and disappear on pressure. Later in the disease an eruption of minute transparent vesicles *(sudamina)* may appear. The *mortality* is about 18 per cent. in hospital and 10 to 12 per cent. in private practice.

RELAPSING FEVER—FEBRIS RECURRENS.

Incubation, 5 to 7 days. Prodromal stage usually absent. Begins with a chill and sudden rise of temperature. Continuous fever 5 to 7 days, and then termination by crisis. Following crisis no fever (*apyrexia*) from 5 to 8 days; after this the temperature again rises as at first, but is of shorter duration. After a period of 7 days there may be a third attack, lasting however from 2 to 3 days only. *Diagnosis*: Enlargement of the spleen, and the presence in the blood of spirilla (*see Blood*). The *mortality* in private practice is about 20 per cent.

MALARIA—FEBRIS INTERMITTENS.

Incubation, 7 to 21 days. Prodromal stage not marked. There is a *chill* followed by rapid rise of temperature lasting but a few hours, and terminating by crisis with profuse perspiration. The period between the termination of one attack and the beginning of another is called *intermission* or *apyrexia*. When the fever recurs every day it is called *quotidian*; every second day *tertian* and every third day *quartan* intermittent fever. Two attacks of fever occurring on the same day is spoken of as *febris intermittens duplicata*. When the second attack of fever occurs at an earlier

hour of the day than the first attack, we speak of *febris intermittens anteponens*, when at a later hour, *post-ponens*.

Fig. 3. Temperature Chart in Febris Intermittens.

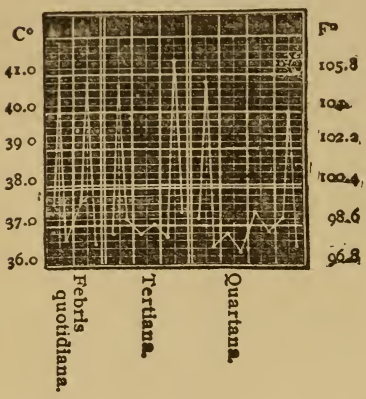
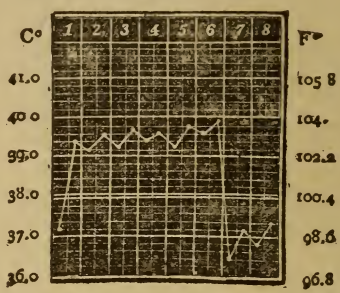


Fig. 4. Temperature Chart in Croupous Pneumonia.



Diagnosis.—Periodicity of the febrile attacks, enlargement of the spleen, specific action of quinine and the *plasmodium malariæ* in the blood. When an intermittent fever does not yield to quinine, *endocarditis*, *latent tuberculosis* or *pus* somewhere in the organism may be suspected.

PNEUMONIA CROUPOSA.

Begins with a severe chill and sudden elevation of temperature. The fever is continuous and ends by crisis on the 3d, 5th, 7th or 9th day. When the fever persists, *empyema* or the termination of the pneumonia in *abscess of the lung*, *gangrene* or *tuberculosis* may be suspected.

Diagnosis.—Dullness, bronchial respiration, crepitant rales, rusty sputum and pneumococci. *Mortality* 8 to 20 per cent. Pneumonias in drunkards are grave.

ERYSIPELAS.

Incubation, 1 to 8 days. Begins with a chill and high temperature. Inflammation of the skin on the 1st or

2d day. Continuous fever during the time erysipelas spreads. Temperature falls when spreading ceases.

Complications.—Œdema of the glottis, bronchitis, pneumonia, endocarditis and cerebral erysipelas (extends to brain from facial vein).

Acute Articular Rheumatism.—The temperature remains steady after the symptoms develop, with evening exacerbations and morning remissions when the joint affection is yielding, but rises when new joints become involved.

Diagnosis.—Tumefaction, redness and tenderness of the joints. Affection yields to salicylic acid, salol or antipyrin.

Complications.—Pericarditis (usually between the 4th and 14th day), endocarditis (in about 20 per cent. of the cases), bronchitis, pleuritis, etc.

Diphtheria.—Temperature is atypical and for the prognosis of little value.

Diagnosis.—A white or gravish exudation on the tonsils, uvula and soft palate. Mucous membrane of nose, larynx or bronchi may be involved in severe cases. Exudation which is not easily removed leaves a bleeding raw surface which is soon covered with a new exudation, Submaxillary and cervical glands enlarged and tender. Weakness and prostration are prominent symptoms.

Sequelæ.—Profound anæmia, nephritis and post diphtheritic paralyses (about 2 to 3 weeks after recovery).

Prognosis.—Always grave. In a mild epidemic average *mortality* 5 per cent., in a severe epidemic 33 per cent.

Acute Miliary Tuberculosis.—Temperature is atypical, although the *typus inversus* is frequent.

Diagnosis.—Cyanosis, dyspnœa, crepitant râles without dullness on percussion and choroid tubercles seen with ophthalmoscope.

Cerebro-spinal Meningitis.—Temperature may be continuous or remittent, and is of long duration.

Diagnosis.—Somnolence, stiffness of the neck muscles, vomiting, retracted abdomen, slow pulse, pupils usually contracted, eruption (absent in one-half the cases) consists of petechial spots of extravasated blood.

Prognosis.—The mortality varying with the epidemic is from 20 to 75 per cent. The disease is less fatal in children than adults.

Syphilis.—Incubation (3 to 4 weeks). *Primary stage;* chancre and swelling of neighboring lymph glands. *Secondary stage* (9 to 11 weeks after exposure to infection); usually begins with a chill and fever (*fever of eruption*) of a remittent type. *Tertiary stage*; development of gummata.

Diagnosis of the Exanthema. Pain and itching not troublesome, copper color, polymorphism (papules macules, pustules, scales, etc. co-exist), tendency to circular form of the patches and eruption shows a predilection for certain parts; around the forehead *(corona veneris)* palm of the hand, sole of the foot, etc.

CHAPTER III.

EXAMINATION OF THE RESPIRATORY SYSTEM.

EXAMINATION OF THE NOSE AND LARYNX.

THE NOSE.

Rhinoscopy. This is the art of inspecting the nasal cavities and naso-pharyngeal space, it is divided into anterior and posterior rhinoscopy. In *anterior rhinoscopy* the view obtained even under favorable conditions is limited, and usually comprises the anterior portions of the lower and middle turbinated bones, with the cartilaginous portion of the septum.

Posterior Rhinoscopy. The rhinoscopic image is made up of the following: vomer or nasal septum, floor of nose, superior meatus, middle meatus, superior turbinated bone, middle turbinated bone, inferior turbinated bone, pharyngeal orifice of Eustachian tube, upper portion of Rosenmüller's groove, glandular tissue at vault of pharynx and posterior surface of velum.

The *nasal cavities* are in direct communication with other cavities situated in the bones of the skull; these are the *antra of Highmore*, situated in the body of the superior maxillary bone and communicating with the nasal cavities by an opening in the middle meatus; *frontal sinuses* situated between the two tables of the frontal bone with an opening in middle meatus, and finally the *sphenoidal cells* or *sinuses*, situated in the body of the sphenoid bone with small openings in the superior meatus. Examination of the nasal cavities is absolutely necessary in diagnosis as many neuroses owe their origin to nasal anomalies, this is notably the case in *asthma* where the paroxysms are found to be associated with nasal polypi and with catarrh of the naso-pharyngeal mucous membrane. "*Running from the nose,*" is a symptom during the invasion of *measles.* Fetor from nose (*ozæna*) may be distinguished from fetor due to

lung gangrene, carious teeth, etc., by testing the breath while the mouth and nostrils are closed alternately. Difficulty in breathing through the nose in infancy (*snuffles*) may be due to syphilis. In bleeding from the nose (*epistaxis*), it must be remembered that the blood may be swallowed or accumulate in the throat and thus simulate *hæmatemesis* or *hæmoptysis*.

THE LARYNX.

Anatomy and Physiology. The larynx is situated between the upper border of the 3d, and lower border of the 6th, cervical vertebra. During respiration, phonation and deglutition, it rises and falls. In *stenosis of the larynx* the rise and fall are exaggerated. *Widening of the vocal chink* (abduction of vocal cords) is effected by the *posterior crico-arytenoid* muscle which also turns the processus vocalis of the arytenoid cartilage outward. *Closure of the vocal cords* (adduction of vocal cords) is effected by the *lateral crico-arytenoid* and *inter-arytenoid* (transverse and oblique) muscles. *Tension of the vocal cords* is maintained by the *crico-thyroid* and *thyro-arytenoid* muscles, the actual muscles of the vocal cords. The *nerve supply* is from the *vagus* with motor branches from the *accessorius*. The *superior laryngeal nerve* innervates the crico-thyroid muscle and muscles of epiglottis (*motor fibres*). It also supplies the mucous membrane of the larynx (*sensory fibres*). The *inferior laryngeal nerve* (recurrent laryngeal) supplies all the other muscles of the larynx not supplied by the *superior laryngeal nerve*. This nerve curves backward around the subclavian artery on the right side and around the arch of the aorta on the left side and passes upward in the groove between the trachea and œsophagus, entering the larynx behind the articulation of the inferior cornu of the thyroid cartilage with the cricoid.

Laryngoscopy.—An examination with the laryngoscope reveals in a normal case the following structures: 1. Epiglottis. 2. Glosso-epiglottic ligaments which connect tongue with epiglottis. 3. Ary-epiglottic folds with the cartilages of Wrisberg. 4. Arytenoid cartilages, cartilage of Santorini, sinus Morgagni. 5. True and false vocal cords, the latter are parallel to and above the former. The true vocal cords are divided into two parts, the anterior part (*ligamentous*) extends to the apex of the processus vocalis, the posterior part, from the apex to the base of same. The anterior part of the glottis is called *Glottis phonatoria*, the posterior part, *Glottis respiratoria*. The laryngeal image, being a reflected one, it is reversed.

Auto-Laryngoscopy.—The use of this method by the student will enable him to attain proficiency in laryngoscopy quicker than by any other method of practice. Let the student seat himself beside a table upon which, at his left, is placed a lamp a little behind his head, and the center of the flame on a level with his eyes. In front of him is fixed an ordinary laryngeal reflector held in some kind of stem, and side by side with it a small toilet mirror. The light from the lamp is reflected on the fauces, the protruded tongue is grasped between the folds of a towel and the laryngeal mirror introduced in the usual manner and the image is seen in the toilet mirror.

The laryngeal mucous membrane is *pale in anæmia*; *red* in acute and *grayish-red* in *chronic laryngitis*. Swelling of the laryngeal tissues occurs in catarrh, œdema and deep-seated inflammation. *Ulcers* may be caused by catarrh (rare), tuberculosis, syphilis, carcinoma and lupus. Exudations, cicatrices, tumors and paralysis of the vocal cord may also be detected by the laryngoscope.

Voice.—1. *Open and closed nasal voice.* The former occurs when closing of the posterior nares is impossible, as in paralysis or ulcerative destruction of the soft palate. The latter occurs in obstruction of the nose, e. g. polypi and catarrh. 2. *Hoarse voice*, in various affections of the larynx. 3. *Want of voice* (aphonia) in functional and organic affections of the larynx. *Intermittent aphonia* is usually hysterical, it begins and disappears suddenly, and the cough may be clear. 4. *Bass voice*, in destruction of the vocal cords. 5. *Diphthonia* in polypi of the vocal cords.

PARALYSIS OF THE VOCAL CORDS

During inspiration the true vocal cords separate, coming together again during expiration. In the act of singing the vocal cords come in almost immediate contact; and in laughing and coughing they intermittingly strike against each other.

Paralysis of the Recurrent Laryngeal. Leads to paralysis of the muscles supplied by this nerve. If double-sided (rare), the vocal cords are immovable in the half-way position in phonation and respiration. When the nerve is paralyzed on one side the healthy vocal cord in respiration moves outward, while in phonation it approaches the affected cord by crossing of the arytenoid cartilages.

Symptoms. Aphonia without dyspnœa.

Paralysis of Individual Branches.—*Paralysis of the Posterior Crico-arytenoid Muscle.* The vocal cord can not be moved outward in respiration. In paralysis of

both cords there is only a narrow space for air to enter the larynx.

Symptom. Pronounced inspiratory dyspnœa.

Paralysis of the Inter-arytenoid. In phonation there remains an open triangle in the posterior part of the glottis.

Paralysis of the Thyro-arytenoid. The vocal cord during phonation is not sufficiently tense, and it is bowed outward with its free edge concave.

Fig. 5.

Fig. 5. a. Paralysis of the crico-arytænoideus posticus; position of inspiration. b. Paralysis of the inter-arytænoideus; phonation. c. Paralysis of the thyro arytænoideus; phonation. d. Paralysis recurrent laryngeal on both sides; respiration and phonation. e. Paralysis of the thyro-arytænoidei and inter-arytænoidei muscles.

Paralysis of the Adductors (lateral crico-arytenoid and inter-arytenoid). The glottis remains open during phonation as a large triangle. Paralysis of the lateral crico-arytenoid alone, gives the glottis a lozenge shape.

Paralysis of the Superior Laryngeal Nerve. The voice is deeper because the crico-thyroid muscles which render the vocal cords tense are paralyzed. When the finger is placed between the thyroid and cricoid cartilages they are no longer approximated, as is the case normally when an attempt is made to produce high tones. There is also *anæsthesia* of the laryngeal mucous membrane extending down to the vocal cords.

Paralysis of the muscles of the larynx may be *myopathic* or *neuropathic*, according to whether the paralysis is due to affections of the muscles or nerves. According to the functions of the muscles of the vocal cords, we have paralysis of *respiration*,

phonation and *combined paralysis.* The *posterior crico-arytenoid* muscles are concerned in the first form, their object being to separate the vocal cords during inspiration, thus allowing air to enter the lungs. All the other laryngeal muscles are concerned in phonation and their involvement leads to paralysis of phonation, whereas a combination of both conduces to combined paralysis.

The following are some of the causes of paralysis: 1. Affections of the *central nervous system* (lesions of the medulla, pons and cerebrum). 2. *Compression of the vagus* or its branches (tumors, aneurism, pericarditis, pleuritis, enlarged bronchial glands, etc). 3. *Neuroses* (hysteria and epilepsy). 4. *Reflex paralysis.* 5. *Toxic* (lead, opium, belladonna, etc). 6. *Infectious diseases* (diphtheria, typhoid fever, variola, etc). 7. *Diseases of the larynx.*

Palpation, Percussion and Auscultation of Larynx.—If the fingers are placed lightly on the larynx while speaking a peculiar vibration (*laryngeal fremitus*) is communicated to them, which is equally strong on both sides. In laryngeal paralysis, laryngeal fremitus is diminished or absent. *Internal palpation of larnyx* is of great value in determining the presence of foreign bodies and œdema of the glottis. *Percussion of the larynx* gives a *tympanitic sound, auscultation* gives loud tubal respiration, called *laryngeal respiration.*

DISEASES OF THE LARYNX.

Acute Laryngitis.—*Laryngoscopical examination.* Hyperæmia (diffused or circumscribed) swelling of mucous membrane, and at times superficial erosions.

Diagnosis. Cough, expectoration, hoarseness, aphonia, tickling in the throat and slight pain in swallowing.

Chronic Laryngitis.—*Laryngoscopical examination.* Mucous membrane of a bluish-red color, and thickening of affected parts. Vocal cords occupied by nodular eminences. (*Chorditis tuberosa*). Erosions.

Diagnosis. Voice readily fatigued and from hoarseness it may pass over to aphonia, slight cough and expectoration and morbid sensations in the larynx (pressure, dryness, tickling, etc).

Laryngitis Diphtheritica.—*(Laryngeal croup). Laryngoscopical Examination* (opportunities are rare). Fibrinous exudation.

Diagnosis. Stenosis of the larynx (in—and expiratory dyspnœa and excessive activity of the respiratory mus-

cles), barking, brassy cough, cyanosis and stupor *(carbonic acid intoxication)*. Fever usually moderate, unless inflammation extends to trachea and bronchi.

A distinction must be made between *primary* and *diphtheritic* croup which resemble each other.

Primary Croup is. 1. A local disease. 2. Begins in larynx. 3. Pharynx slightly affected. 4. Neither contagious nor infectious. 5. Not epidemic.

Diphtheritic Croup is, 1. A constitutional disease. 2. Begins in the fauces. 3. Pharynx extensively affected. 4. Contagious and infectious. 5. Epidemic.

False Croup (catarrhal laryngitis) may be confounded with *true croup* (croupous laryngitis). The former begins suddenly in perfect health, whereas in true croup, cough, hoarseness, fever and angina precede the attack. Dyspnœa when present in false croup is of short duration, and we find no diphtheritic throat deposits.

Œdema of the Glottis.—*Laryngeal examination.* Swelling of the mucous and sub-mucous tissues, especially marked in the epiglottis and ary-epiglottic folds.

Diagnosis. Dyspnœa is very great and very sudden. First it is inspiratory, but soon becomes inspiratory and expiratory. Swelling is distinctly felt by the examining finger.

Laryngeal Tuberculosis.—Occurs as a complication in phthisis pulmonalis in about 30 per cent. of the cases.

Laryngoscopical Examination. The first manifestation is pyriform thickening of the mucous membrane covering arytenoid cartilages. Later, *tubercles* are seen in the mucous membrane as small yellowish-white spots (*second stage*). The *third stage* is the stage of fully developed ulceration. *Phthisical ulcers* are broad, shallow, irregular, gray in color, and essentially of slow progress. The exudation from the ulcers contains the tubercle bacillus.

Diagnosis. Fever, pulmonary tuberculosis, hoarseness, aphonia, pain in deglutition and demonstration of the bacilli.

Laryngeal Syphilis.—*Laryngoscopical examination.* In the early stages the mucous membrane is of a rose-red color *(syphilitic laryngitis)*. Later stages, circumscribed

infiltrations or diffuse gummy deposits which rapidly degenerate (characteristic) leaving ulcers, which are deeply excavated with sharp cut edges, yellow purulent discharge, and rapidly destructive.

Syphilitic ulcers are frequently found on the epiglottis.

Diagnosis. History of syphilis and other symptoms of this affection. Successful results from antisyphilitic treatment. Syphilitic affections of the larynx are usually painless, and may result in dangerous adhesions, cicatrizations and stenosis.

CHAPTER IV.

EXAMINATION OF THE THORAX.

Topography of the Chest.—For localizing disease and defining the situation of organs, the chest is regionally divided as follows:

Anterior regions.—1. *Supra-clavicular*, triangular shaped space above the clavicle containing the apex of lung which rises from ½ to 1½ inches above the clavicle. 2. *Infra-clavicular*, extends from clavicle to 3d rib. 3. *Supra-sternal* (jugular fossa), hollow space above notch of sternum and bounded on either side by the sterno-cleido-mastoid muscle. 4. *Sternal*, occupied by the sternum.

Lateral Regions.—1. *Axillary*, bounded by the anterior and posterior axillary lines.

Posterior Regions.—1. *Supra-scapular*, situated above scapula and contains the apex of lung which rises to 7th cervical vertebra. 2. *Infra-scapular*, below scapula. 3. *Inter-scapular*, between the scapulæ and divided into right and left by vertebral column. Contain on both sides; lung, bronchi and bronchial glands; opposite 3d dorsal vertebra (2d rib in front), bifurcation of trachea occurs. Left inter-scapular region also contains œsophagus, and from 4th dorsal vertebra downward, the descending aorta.

When exactness is required in localization, measurements may be taken from definite anatomical landmarks.

The breadth of thorax is determined by the following perpendicular lines:

1. *Median*, drawn through the middle of sternum.
2. *Sternal* (right and left), drawn along borders of sternum.

3. *Mammary*, drawn through nipple which lies usually in the 4th inter-costal space 4 inches from the sternal border.

4. *Parasternal*, between mammary and sternal lines.

5. *Anterior Axillary*, from the lower border of pectoralis major.

6. *Posterior Axillary*, from lower border of latissimus dorsi.

7. *Middle Axillary*, between anterior and posterior axillary lines.

8. *Scapular*, from inferior angle of scapula.

9. *Costo-articular Line*, from sterno-clavicular articulation to tip of 11th rib.

Landmarks.—*Top of sternum* is on a plane with the 2d dorsal vertebra. *Angle of Louis* is a bony prominence at the union of manubrium with the body of the sternum and represents the 2d rib (useful in counting the ribs). *Sibson's furrow* is at the lower border of pectoralis major. *Harrison's furrow* is at the level of xiphoid process and corresponds to normal attachment of the diaphragm. *Inferior angle of scapula* is situated at about the 7th rib. The *Fossa of Mohrenheim*, is a depression under the clavicle in the outer part of the infra-clavicular region between the pectoralis major and deltoid muscles.

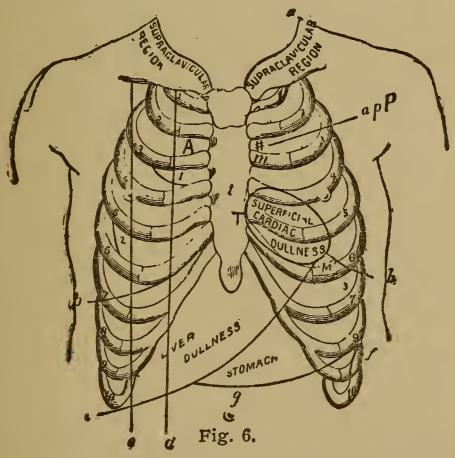


Fig. 6.

Fig. 6. a. Upper boundary of right and left lung. b. Lower boundary of right and left lung and upper border of absolute liver dullness on right side. c. Lower boundary of hepatic dullness. d. Parasternal line. e. Mammary line. f. Splenic dullness.

g. Lower boundary of distended stomach. M. Auscultation of mitral valve. m. Anatomical position of mitral valve. A. Auscultation of aortic valves. T. Auscultation of tricuspid valve. t. Anatomical position of tricuspid valve, ✻ a p P. Anatomical position of aortic and pulmonary valves and auscultation of latter. 1. Right superior interlobar fissure, showing boundary line between the upper and middle lobes of right lung. 2. Right inferior interlobar fissure. 3. Left interlobar fissure ending in the mammary line at the 7th rib.

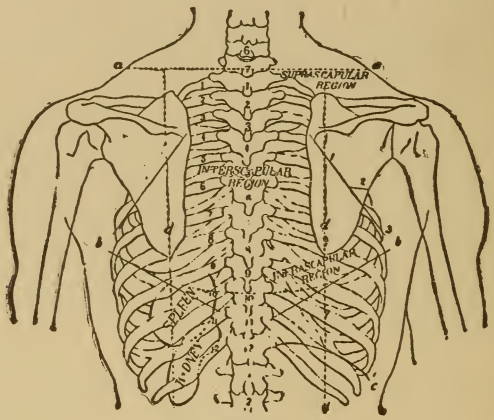


Fig. 7.

Fig. 7. a. Upper boundary of right and left lung. b. Lower boundary of right and left lung. c. Lower boundary of liver. d. Scapular line. 1. Right and left interlobar fissures; the former dividing into superior (2) and inferior (3) interlobar fissures.

Methods of Physical Diagnosis.—1. *Inspection.* 2. *Palpation.* 3. *Mensuration.* 4. *Percussion.* 5. *Auscultation.* 6. *Succussion.*

Phonometry, *plegaphony* and *thermo-palpation* are auxiliary methods of diagnosis. Phonometry is a method of examination consisting in the application of a tuning-fork to determine the physical condition of the thoracic organs. When the handle of a tuning-fork after being struck is applied successively to the thigh, stomach and thorax, the tone with reference to duration and intensity is different in all three situations. Over the stomach the greatest and over the thigh the least resonance is obtained; while over the lung the resonance is weak. In relaxation of the lung and over lung cavities the resonance is loud, whereas over

consolidations it is weak. Plegaphony is introduced as a substitute for *bronchophony* when the latter can not be practiced from any cause which prevents the vibration of the vocal cords as in diseases of the larynx. In this method of examination, the larynx is percussed, the observer auscultating the chest wall at the same time. The percussion tone in the larynx is conducted by the air in the bronchi to the chest wall. Over infiltrated lung, the sound loses but little of its original character. Over an exudation in the pleura, the sound is weakened or absent. Over cavities the sound is loud and tympanitic. Thermo-palpation is a new method of examination described by Benczur and Jonas. The principle governing it is that the skin, when palpated over air containing organs, is warmer than over airless viscera. Thus, palpation with the palmar surface of the finger shows a diminished temperature over the regions occupied by the liver, heart and spleen, when compared with adjacent parts. It is also recommended in defining pathological processes; the upper border of pleural exudations, and the borders of aneurisms and abdominal tumors are said to be accurately determined.

INSPECTION.

This signifies observation of the chest. We determine by inspection: 1. *Form of Thorax.* 2. *Thoracic movements.* 3. *Frequency of respiration.* The normal chest is nearly symmetrical. Not over 20 per cent. of people have a perfectly symmetrical chest. The anterior walls of a normal chest arch forward to the nipples and then slope downward to the lower ribs. Even in health, the supra-clavicular regions are slightly concave.

The most common deviations of a perfect chest are slight curvatures of the spinal column. This column has a normal curvature at the cervical, dorsal, lumbar and sacral regions. A curvature of the column convexly backward is called *Kyphosis*; a curvature forward, *Lordosis*; a lateral curvature, *Scoliosis*; and a curvature laterally and backward, *Kypho-scoliosis.* Scoliosis frequently results from contraction of one lung or chronic changes in the pleura.

Pathological Changes in Shape.—1. *General bilateral bulging* (barrel-shaped chest) occurs in Emphysema.

2. *Unilateral dilatation* is observed in pleural exudations, pneumothorax and croupous pneumonia.

3. *Circumscript bulging* in emphysema, encapsulated exudations, tumors and enlargement of viscera.

4. *Paralytic Thorax* is characteristic of pulmonary tuberculosis. Chest is flat, narrow and long; intercostal spaces wide, bony structures prominent, and the scapular angles project like wings.

5. *Unilateral retraction* occurs after pleural exudations and contraction of lung.

Retraction after pleural exudations is explained as follows: The lung compressed by fluid and bound by adhesions does not expand to its previous dimensions after absorption of the fluid, thus allowing the atmospheric pressure to produce retraction of the chest on the affected side.

6. *Circumscribed retraction.* After retraction of a cavity in lung. Observed in supra-clavicular region, it may indicate shrinkage of the lung apex. The supra-clavicular regions are normally depressed, and it is only when the depression is exaggerated, or more marked on one side, that apical disease is suspected.

7. *Chicken Breast* (pectus carinatum). The sides of the chest are flattened and the sternum has a keel shape. Observed in children when unusual force is exerted upon lungs.

8. *Rickety Chest.* A shallow, longitudinal groove is found on either side of the chest, at the junction of the ribs and costal cartilages.

9. *Funnel Breast.* Depression at lower part of sternum. Congenital.

10. *Cobbler's Breast.* Like former, although acquired, and is due to the pressure of instruments against the breast.

Transverse Constriction of the Chest. A well marked depression of the lower portion of chest wall in front, from the xiphoid cartilage to the axillary line is due to some impediment to the free entrance of air into the lower portion of the lungs. It is usually present in *bronchitis* during infancy.

Thoracic Movements.—In normal respiration, inspiration is an active and expiration a passive act. The lungs undergo no active movements during respiration but passively follow the movements of the thorax and diaphragm.

Important Muscles of Inspiration. Intercostal muscles and diaphragm. In females, the scaleni muscles are normally active as muscles of inspiration.

Accessory Muscles of Inspiration. Sterno-mastoid, scaleni, pectorales, serrati and trapezius. These muscles are only employed when the blood is overloaded with carbonic acid gas, causing difficulty of breathing (dyspnœa).

Muscles of Expiration. Abdominal muscles, quadratus lumborum and serratus posticus inf.

The act of *expiration* is caused normally by the elasticity of the chest unaided by muscles. *Inspiration* is accomplished by the elevation of ribs and sternum, and the rotation outward of the former by the external and internal intercostal muscles. This constitutes the *costal type of respiration.* Inspiration is furthermore aided by contraction of the diaphragm (*abdominal type of respiration*). The type of respiration in *males* is a combination of costal and abdominal (*Costo-abdominal respiration*). In *females* and *children* it is costal. The *thoracic movements* are usually equal on both sides. Sibson demonstrated that the right expands more than the left side of thorax which Eichhorst attributes to the better developed muscles on the right side, increased width of right bronchus and larger size of right lung. The connection between sex and type of respiration has been variously discussed. It is maintained by some that the diaphragm is the essential muscle of respiration, but that its movements are impeded in women by the wearing of corsets, this necessitating the vicarious action of the intercostal muscles. Hutchinson maintains that the costal type is present in girls who have never worn corsets, and explains the costal respiration in females by supposing that pregnancy would interfere with the movements of the diaphragm. Perhaps the greater elasticity of the thorax in women and children is the cause of the costal type.

Pathological Changes in Thoracic Movements.—*Costal Respiration in Males* occurs in any interference with the movements of the diaphragm (pain, mechanical obstruction and paralysis). *Abdominal Respiration in Females* occurs in paralysis of the inspiratory muscles and in thoracic pain.

The action of the diaphragm can be noted by a prominence of the epigastrium occurring during inspiration.

Asymmetry of Respiration is observed in painful thoracic affections, and in diseases of the thoracic viscera. *Inspiratory Retraction of Chest* indicates that air does not

enter lung alveoli, which allows the external atmospheric pressure to preponderate.

In the lower lateral portions of thorax, *physiological retractions* are often present, but occurring as they do only during the beginning of inspiration, they are diagnosed from *pathological retractions* which persist during the entire act.

Diffuse Unilateral Retraction, in obstruction of the main bronchus. *Expiratory Bulging of Chest*, in emphysema and phthisis. It can only occur when positive intrathoracic pressure exists.

Frequency of Respiration.—In the *adult male* there are 16 to 24 respirations per minute. The normal relation between *frequency of respiration and pulse*, is as 1:3½ to 4. It takes four times as long for the blood to go through the systemic as through the pulmonic circulation.

The respirations are more frequent in females. *Influence of age* on the number of respirations: at birth, 44; 15th to 20th year, 20; 20th to 25th, 18; 25th to 30th; 16; 30th to 50th, 18 times per minute. *Influence of position on the number of respirations:* lying, 13; sitting, 19; standing, 22 times per minute. During *sleep*, the number of respirations is diminished.

The *respirations* are *diminished* in frequency in cerebral affections, infectious diseases, opium poisoning, and in stenosis of the air passages.

When the seat of obstruction is in the larynx, at every inspiration the latter descends, to rise again during expiration; whereas, in obstruction below the larynx, the latter is comparatively immovable.

The *respirations* are *increased* in frequency in painful affections, e. g. pleuritis, rheumatism of the thoracic muscles, peritonitis, accumulation of carbonic acid in the blood, and from nervous causes, e. g. hysteria.

An increase in the frequency of respiration also occurs in *fever*, and is due to the direct action of the heated blood on the center of respiration in the medulla. In dyspnœic persons, the difficulty in counting the respirations may be overcome by placing the finger on the *scaleni muscles* (situated in the neck, between the trapezius and sterno-mastoid muscles), which at every inspiration are raised by their contraction. By counting the elevations of the finger, the number of respirations is determined.

The *respirations* are *irregular* in coma and Cheyne-Stokes' respiration.

Cheyne-Stokes' respiration is a form of dyspnœa in which periods of complete cessation from breathing (*apnœa*) are varied with periods of slowly rising respiratory movements, which, after becoming gradually deeper, become slower and shallower, until they cease altogether. Observed in diseases of the brain and heart, coma and opium poisoning, although it may occur in health during sleep.

Dyspnœa (*difficulty of breathing*), occurs when blood is surcharged with carbonic acid gas. The *forms of dyspnœa* are inspiratory, expiratory, or a combination of both. *Inspiratory dyspnœa* occurs in paralysis of the crico-arytenoidei postici, which in health open glottis; and in narrowing of the air passages. *Expiratory dyspnœa* is observed in bronchial asthma and emphysema, and when movable tumors so situated below the glottis only occlude latter during expiration.

Respiration is regulated by the nervous system and by stimulation of the respiratory center in the medulla. As long as the oxygen is maintained at a certain standard in the blood, this center is not stimulated, but whenever the oxygen is reduced, stimulation of the center, characterized by an increase in the number of respirations, results.

PALPATION.

This is an adjunct to inspection, and is the act of laying on the hand. The objects determined are; *pain, movements of thorax, palpation of vocal, bronchial,* and *friction fremitus* and *fluctuation.*

Pain on Pressure is observed in visceral affections involving the pleura, and in painful affections of chest-wall. In the diagnosis of respiratory diseases, the important fact must be remembered, that the development of pain nearly always indicates inflammatory involvement of the pleura. In *intercostal neuralgia*, three painful points (*points douloureux*) are usually detected (vertebral, lateral, and sternal points). In *rheumatism of the thoracic muscles*, the pain is diffused, and the affected muscles are painful when compressed by the fingers. In practicing *palpation of the thoracic movements*, place the hands on symmetrical parts of the thorax. In *pneumonia, pleuritis,* and *unilateral tuberculosis*, the thorax over affected parts is retarded in action. The *action of the diaphragm* is determined by placing both fingers in the epigastric region on either side of the median line. An absence of diaphragmatic contraction on one side is present in *diaphragmatic pleuritis, local peritonitis,* and *paralysis of phrenic nerve.*

Vocal Fremitus is the vibration of the thoracic walls during speaking, perceptible to the hand. It is due to the vibration of the vocal cords in speaking communicated by the column of air in trachea and bronchi to the air vesicles, and so to the chest-wall. It is more evident on the right side of thorax, because the right bronchus is more capacious and given off at a less acute angle than the left. Vocal fremitus is diminished as the distance from larynx increases.

The louder and deeper the voice, the more evident the *vocal fremitus*. For this reason it is more evident in males than females, while in children it is with difficulty felt. In determining vocal fremitus, the palmar surface of hand is applied to chest. If greater accuracy is required in limiting the fremitus, use *linear palpation*, which consists in placing one end of a rod of wood (a lead pencil will suffice) on the chest, and supporting the other end with the fingers. In this way the vibrations of chest-wall are well conducted from a limited area. In limiting the situation of solid viscera (liver, spleen, heart,) from the lung, *linear palpation* is well adapted, the fremitus being absent over the airless organs. *Vocal fremitus is increased* in consolidation of the lung (better conduction), over cavities with dense walls communicating with a bronchus, and in emaciated persons. *Vocal fremitus is diminished, or absent*, in pleural exudations, pneumothorax, obstruction to bronchus, and in excessive development of chest coverings (fat and muscles). The student will do well to repeat the following experiments: Remove from the cadaver, the lungs and trachea. Into latter tie a rubber tube, into the end of which introduce a funnel. When the latter is spoken into, the hand can readily feel the fremitus over the lung. Next, inflate a stomach, and placing it over the lung, feel for the fremitus, it is absent (analogous condition in pneumothorax). Now fill a rubber balloon, or similar object, with water, and placing it over the lung, feel for fremitus, it is also absent (analogous condition in pleural exudations).

Bronchial Fremitus is sometimes present in bronchitis. In this affection the air in the tubes is thrown into extra vibration, by the vibration of the bronchial walls and of the fluid contained within them.

Friction Fremitus (*pleural fremitus*) is the grating of one roughened surface of the pleura over the other.

Each lung is covered by a serous membrane (*visceral layer of pleura*), which is reflected upon the inner surface of the thorax (*parietal layer*). Both layers are brought in constant contact by the movements of respiration, but during health they are smooth,

and no friction results. In inflammation the fibrinous exudation conduces to friction fremitus. *Fluctuation* is present in excessive pleural effusion. Palpation may determine the extra or intra-pleural origin of pus accumulation. If the *abscess* is intra-pleural, gradual pressure will cause its disappearance; not so, if the abscess is of extra-pleural origin.

MENSURATION.

The *circumference* of the chest at the height of the nipple in healthy men is about 32½ inches after deep expiration and 36 inches after deep inspiration. The average difference after full inspiration and forced expiration is from 2½ to 3½ inches. The *Sterno-vertebral diameter* measures in healthy men, 6½ inches above and 7½ inches below. The *broad diameter* in men at the height of nipple is 10 inches. The right side of chest exceeds the left by about ¾ of an inch in circumference in right-handed persons. The relative length of inspiration and expiration may be represented as follows: Inspiration, 5; expiration, 4; rest, 1.

In *emphysema* expiration is three times as long as inspiration. In taking the above measurements a graduated tape is all that is necessary. Select the spinous process of a vertebra behind and the median line in front as fixed points of measurement. The sterno-vertebral and transverse diameters are determined by means of callipers.

Stethometry is a method of graphically recording the movements of the chest by means of the stethometer.

Pneumatometry is a method of measuring the force of inspiration and expiration by means of a mercurial manometer (*Pneumatometer*). It shows that the power exerted in expiration is greater than in inspiration. The inspiratory power is diminished in phthisis, stenosis of the air passages and pneumonia. The expiratory power is diminished in emphysema, asthma and bronchitis.

Spirometry is a method of measuring the amount of air received into the lungs by means of the spirometer.

The *total capacity of the lungs* in men is about 200 to 250 cubic inches, in women 163 cubic inches. This capacity increases with the growth of the body. *Tidal air* is that which goes in and out in ordinary respiration, and is about 33 cubic inches. *Reserve air* is

the air which after quiet expiration can be expelled by forced expiration, and is about 100 cubic inches. *Complemental air* is the air which after quiet inspiration can be introduced by forced inspiration, and is about 100 cubic inches. *Residual air* is the air which remains in the lungs after the deepest expiration, and is equal to about 100 to 125 cubic inches.

PERCUSSION.

This method of diagnosis consists in striking the chest walls to elicit sound. It was first used by Auenbrugger (born 1722, died 1809), a Viennese physician, in 1761. Percussion may be immediate or mediate. *Immediate Percussion* is striking chest wall directly, and is only practiced over clavicle or sternum. *Mediate Percussion* is striking the chest wall indirectly by interposing media. The medium to receive blow is called *pleximeter*, and may be of ivory, glass, wood, leather, metal or the finger. For percussing a hammer (*plessor*) or finger is used. Methods: 1. *Finger—Finger Percussion.* 2. *Finger—Pleximeter Percussion.* 3. *Hammer—Pleximeter Percussion.*

It is better to rely upon the fingers in percussion. Instrumental is easier than finger percussion, although less reliable. The index or second finger of the left hand is applied closely and evenly to the chest, and then tapped with the second finger of right hand. A single, double, or repeated percussion blows may then be made. In *finger—finger percussion* we can appreciate the resistance of tissues percussed (*palpable percussion*) and adapt the finger to irregularities of the chest.

Observe the following: 1. Press finger firmly against part to be percussed. 2. Movement of percussing hand must spring only from wrist, while forearm is motionless. 3. Blow must be sharp and quick, direct and perpendicular. 4. The finger must be instantly removed after percussion, so as not to interfere with chest vibration. The results obtained by percussion are as much due to the method of execution as to the condition of the tissues. The force of the percussion blow is always secondary to the knack of obtaining full vibration in resonant tissues. The student to gain flexibility of the wrist joint should practice movements of this joint with the arm adducted toward the thorax and the forearm at right angles and motionless. *Hammer—Pleximeter percussion* is used for class demonstration, to elicit sounds from thickly padded parts and for uniformity of stroke.

Symmetrical Percussion is used for comparison between the two sides of the chest. Percuss at symmetrical points, using the same percussion blow. This is useful in determining slight variations in the percussion sound.

Palpable Percussion determines the resistance of parts percussed as perceived by the finger.

Auscultatory Percussion consists in auscultating the chest with the stethoscope while percussion is practiced.

Linear Percussion consists in limiting the percussion blow, *e. g.*, striking on the edge of a coin.

Respiratory Percussion consists in percussing after deep inspiration or prolonged expiration.

In *Emphysema* the vesiculo-tympanitic note is unchanged by percussion during the act of breathing. In cases of fluid in the pleural cavity, a deep inspiration will clearly define the border line between the resonance above and the flat note below. In bronchitis, the clearness of normal resonance may become impaired; all doubt is at once dissipated by directing the patient to make a forced inspiration, when the normal lung resonance is restored. Congestion of the lungs in cardiac disease often yields a slight dullness on percussion leading to the suspicion of consolidation; this will be removed after repeated forced inspirations.

Topographical Percussion consists in defining the situation of viscera by dermography.

Dermography is a method of delineating on the chest with an aniline pencil or, better still, with a pencil made for this purpose (*dermograph*), the limitation of organs obtained by percussion. If it is desirable to observe the resorption of exudations, a permanent line may be made with a stick of nitrate of silver. When permanent records are desired for the results obtained by percussion, schemata of the chest may be used on which may be marked with colored inks, the location of viscera, etc.

Light Percussion is practiced when air-containing tissue is to be carefully delineated from airless structures as in topographical percussion. *Strong Percussion* is employed when airless tissue is in immediate contact with thorax and overlies air-containing tissue and resonance of latter is desired; or to obtain dullness from airless tissue covered by tissue containing air. The *percussion blow is propagated* from 1½ to 2½ inches on the surface and to a depth of about 2½ inches. *Caution.* Every percussion blow is traumatic in action and when often repeated aggravates or may even induce inflammatory affections. Over aneurisms (danger of dislodging thrombus) and painful points, percussion must be light. Patients suffering from *hæmoptysis* should not be percussed.

The sound produced by percussion of the chest possesses *pitch* and *intensity*. Pitch depends on the number of vibrations in a given unit of time. Over anything solid (heart, liver) the pitch is high; over normal lung, the pitch is low. The greater the quantity of air, the lower the pitch. Pitch is highest over fluids because they are most dense. Intensity is the loudness of percussion note and depends chiefly on the force exerted in percussion. *Percussion sounds* may be 1. *Clear*, 2. *Dull*, 3. *Tympanitic*. *Modifications of tympanitic*; 1. *Amphoric* or *metallic*, 2. *Cracked-pot or cracked-metal sound*. The percussion sound is caused by vibration of chest wall and the air within the alveoli of lung.

PERCUSSION OF THE THORAX.

Over the lung like any other air-containing organ percussion yields a clear sound and is called *normal vesicular* or *pulmonary resonance*. Over the liver and heart like other airless viscera, the sound is dull. The left infra-clavicular region is usually taken as the standard of pulmonary resonance. Always percuss symmetrical parts of the thorax, using the same percussion blow. The normal percussion note (*pulmonary resonance*) is compared by Flint to the sound elicited by percussing a loaf of bread covered with a napkin. The posterior thoracic regions do not yield a percussion note of the same intensity as the anterior regions on account of the density of the dorsal muscles. In percussing the *interscapular space*, the patient's head is bowed and the hand on either side is made to grasp the convexity of the shoulder. This position as suggested by Corson will increase the interscapular region more than twofold and will render the dense muscles tense and thin, thus reducing to a minimum obstacles to percussion and auscultation. The percussion note over the apices posteriorly is less intense in right-handed individuals on the right than on the left side (the opposite holds good in left handed individuals) owing to the better developed muscles in the former situation. Similarly the percussion note is less intense on the right side on the anterior surface of the chest.

Dullness over the lung is obtained: 1. When lung adjacent to thorax is deprived of air by infiltration or atelectasis. The airless tissue must be at least 1½ inches in extent, in order to be recognized. *Infiltration of the lung* occurs in pneumonia, phthisis, hæmorrhagic infarction, tumors and abscess. *Atelectasis* results from

compression of the lung *(pleuritic* and *pericardial exudations)* or by an obstruction in the bronchus. 2. When fluid *(pleuritic effusion)* or solid substance (*tumor or thickened pleura*) is between chest and lung. In adults 400 ccm. of fluid at least, must be present in order to be recognized.

Pleuritic Exudations first accumulate in pleural cavity provided no adhesions exist, in the posterior or inferior parts, thence extending, if the fluid increases, forward and upward. If the exudation was formed during the time patient was in bed, then the upper border of dullness will describe a line higher behind than in front. If exudation was formed during the time patient was walking about then the line is almost *horizontal.* In *exudations undergoing resorption,* the upper border often has a curved course, the highest part of which is in the axillary region (*Curve of Ellis*). Inflammatory pleural exudations change little or not at all when the patient changes position, because exudation is encapsuled by adhesions. This observation has recently been confirmed by the investigations of Strauch. *Hydrothorax* (generally bilateral) gives a dullness changeable on position. When air and fluid are present in the pleural cavity (*pyo-and sero-pneumothorax*), a dullness is present in the lower anterior portion of thorax when patient is erect, which is supplanted by a tympanitic sound in the recumbent posture. It will be observed that the finger appreciates an increased sense of resistance on percussion when fluid is present in the pleural cavity. The presence of large quantities of air or fluid in the pleural cavity depresses the diaphragm, enlarges the affected half of thorax and dislocates the heart toward the healthy side. The depression of the diaphragm in exudation of the right pleural sac causes displacement downward of the liver (*see percussion of liver*). In exudations on the left side, the *half-moon shaped space of Traube* disappears. This space is found in normal individuals between the left lobe of liver, lower border of left lung and anterior end of spleen. It extends upwards to the 5th or 6th costal cartilage, its upper border describing a convex line directed upward. It contains the fundus of the stomach and yields a tympanitic percussion note. Beginning resorption of a pleuritic exudation on the left side is first manifested by the reappearance of the tympanitic percussion sound of this space. Emphysema diminishes and contraction of the left lung increases the extent of this space.

A *Tympanitic sound* indicates the presence of air within a cavity with elastic walls. The sound over the stomach is typically tympanitic.

Tympanitic sound over the lung is obtained : 1. In *consolidation of lung* which allows transmission of percussion blow to the air in bronchus and trachea as in con-

solidation of the upper lobe (William's tracheal tone). 2. In *pathological air conducting cavities*, (a) In bronchiectatic or tuberculous cavities, empty of fluid, near the surface and at least as large as a walnut. (b) In *pneumothorax*, when the air is under low tension. Usually, however the sound is loud but not tympanitic.

A *metallic sound* can almost always be obtained in *pneumothorax*, if the following procedure is adopted: fix a plexiметer over the affected side and while one observer is percussing the plexiметer with a lead pencil, another auscultates the thorax and hears a fine metallic sound.

Pyo pneumothorax Subphrenicus is a collection of pus and air between the liver and diaphragm on the right side in consequence of perforation of the stomach or intestines. Such abscesses more rarely occur on the left side. They are diagnosed from pneumothorax as follows: history of preceding peritonitis, absence of cough and expectoration, slight dislocation of the heart only, intercostal spaces not bulged, vesicular respiration heard as far down as the abscess. *Pleuritic friction* can be heard throughout the region of subphrenic dullness. High punctures in the 5th intercostal space show a collection of pus or serum while a low puncture, according to Scheurlen, in the 8th intercostal space, yields a pus which is always *ichorous*.

3. In *relaxation of the lung tissue*, as in the neighborhood of exudations and infiltrations.

4. In *incomplete infiltrations* when lung tissue contains air and fluid, as in *œdema, catarrhal and croupous pneumonia* (1st and 3d stages).

The *Amphoric Sound* is a concentrated tympanitic sound of raised pitch, and denotes a large cavity, with firm, elastic walls. It resembles the sound obtained by percussing the cheek when the mouth is closed and fully but not tensely inflated. It also resembles the sound produced on percussing an empty bottle. It is heard over cavities of a diameter not less than 2½ inches. Also heard in *pneumothorax* when the tension of air is of a certain degree.

Cracked-Pot Sound (*bruit de pot fêlé.*) This may be imitated by striking the back of the hands loosely folded across each other, against the knee; the sound resulting is like the clinking of coin. The conditions necessary for its production seem to be the sudden escape of air from an inclosed space under pressure. A strong percus-

sion blow is necessary. It may be heard in healthy persons, especially children, if the chest is percussed during speaking or crying. *Pathological occurrence:* 1. Over cavities communicating with a bronchus. 2. Over pleural effusions. 3. In pneumonia around consolidated parts. 4. In pneumothorax with opening in the thoracic wall. The cracked-pot sound is clearer when patient opens mouth and protrudes tongue during the act of percussion, or when it is practiced during expiration.

Wintrich's Change of Note is that in which the percussion note is higher when the mouth is open, and lower when it is closed. Usually heard in cavities in free communication with a bronchus. This phenomenon can be imitated by percussing larnyx with the mouth open and then closed.

Wintrich's Interrupted Change of Tone is the preceding phenomenon observed on lying down, and absent on sitting up, or *vice versa*. It indicates that the bronchus leading to the cavity is obstructed in certain positions by the fluid contents.

Respiratory Change of Tone is a higher tone in deeper inspiration, and is heard over cavities.

Gerhardt's Change of Tone, when present, is an almost certain sign of a cavity, and is due to the presence of fluid in a cavity, changing the percussion sound according to the position of the patient.

Biermer's Change of Note is the percussion note in *sero-pneumothorax*. It is deeper in the erect than recumbent posture, because in the former position the fluid displaces the diaphragm downward, thus making the sounding cavity larger.

PERCUSSION OF THE LUNGS.

The *apices* of the lungs extend above the clavicles in the supraclavicular regions from 1–1½ inches; behind they rise in the supra-scapular regions to a level with the spinous process of the 7th cervical vertebra. Resonance of the apices is separated from dullness of surrounding structures by light percussion, but care must be exercised in percussing away from the trachea.

Situation of the Lower Border of Right Lung (Figs. 6 and 7).

In right *sternal* line; upper border of cartilage of 6th rib.
" " *parasternal* " lower " " " " " "
" " *mammary* " upper " " " 7th "
" " *axillary* " lower " 7th rib.
" " *scapular* " at 9th rib.
At *vertebral column* at 11th dorsal vertebra.

Situation of the Lower Border of Left Lung:

In left *mammary* line ; upper border of cartilage of 7th rib.
" " *axillary* " lower " " " " "
" " *scapular* " at 9th rib.
Along vertebral column at 11th dorsal vertebra.

In practicing topographical percussion of the lungs, or other organs, the *percussion blow must be light.* In *children*, the lower border of lung is higher by one intercostal space, whereas in the *old*, it is one intercostal space lower. The lower border of right lung is separated from the liver dullness; the lower border of left lung from the dullness of the spleen and left kidney, and the tympanitic sound of the stomach.

TOPOGRAPHY OF THE LOBES OF THE LUNGS.

The following table will approximately define the situation of the pulmonary lobes on the different surfaces of the thorax. (Figs. 6 and 7.)

Right Lung.

	Anterior Surface.	*Lateral Surface.*	*Posterior Surface.*
Upper lobe.....	Extends to 3d or 4th rib	Extends to 4th rib.	To spine of scapula.
Middle "	From 4th rib downward	4th to 6th rib.	.....
Lower "	.....	6th to 8th rib.	From spine of scapula downward.

Left Lung.

Upper lobe.....	In mammary line to 6th rib	Extends to 4th rib.	To spine of scapula.
Lower "	.....	From 4th rib downward.	From spine of scapula downward.

Dislocation of Lung Border.—During deep inspiration the lower border of the lung descends and ascends during expiration (*active mobility*). During deep inspiration when the patient is lying on the side, the lung may descend as much as 3½ inches. The dislocation of the lower lung border is greatest in the axillary

line. The dislocation upward during forced expiration is slightly less than the dislocation during inspiration. During quiet respiration the difference in the position of the lower lung border is not more than ½ inch. The average dislocation of the lower lung border in the various lines during forced inspiration is as follows: parasternal line, 1 inch; mammary line, 1⅓ inch; axillary line, 1½ inch; scapular line, 1 inch.

Passive mobility. This is observed when the position of patient is changed. In the recumbent, the lower lung border descends about ½ inch lower than in the erect posture. When lying on the side, passive mobility is greatest. Active and passive mobility are *decreased* in emphysema and pleuritis and absent when lower lung border is adherent.

The lower border of the lung is *permanently lower* in emphysema and *temporarily* during asthmatic attacks. It is *higher* than normal *on both sides* when diaphragm is pushed upward (air, fluid and tumors in abdominal cavity). It is *higher on one side* in contraction of the lung or pleura.

The visceral layer of pleura directly covers the lung, whereas the parietal layer forms a cavity in which the lung is allowed considerable movement. This cavity forms spaces in many places which are larger than the volume of the lung, especially at its lower border. These spaces (*complemental or reserve spaces*) allow of an augmentation of the lung, and it is there where fluid in the pleural cavity first accumulates.

AUSCULTATION.

Definition.—Application of the ear to the chest for the detection of sounds. *Methods of auscultation:*—1. Mediate or indirect. 2. Immediate or direct.

Mediate auscultation is practiced with an instrument called a stethoscope and is possessed of the following advantages: 1. Localization of sound. 2. Obviates chest exposure (important in the examination of women) 3. Avoids contact with the bodies of unhealthy persons. 4. Intensification of feeble sounds. 5. Exclusion of extraneous sounds. 6. Auscultation of regions inaccessible to ear, *e. g.*, supra-clavicular regions.

Disadvantages of the Stethoscope: 1. By intensification sounds are modified. 2. The training of the ear is liable to be neglected. 3. The constant use of the stethoscope may impair the hearing for feeble sounds.

Stethoscopes are of various forms and are made of glass, wood, gutta-percha, metal and ivory. *Binaural stethoscopes* are used in preference to others, and the objection that they modify and exaggerate sounds is of minor consideration when compared to their many other advantages.

Students should accustom themselves to the use of one stethoscope only. Stethoscopes although similarly constructed develop certain adventitious sounds, which become gradually excluded by the trained ear. It is a common occurrence for students who borrow instruments to auscultate badly. In the selection of a stethoscope, note its proper adaptation to the ears. A soft rubber cup attached to the pectoral end of the instrument is of value in auscultating emaciated patients, the object being to secure perfect adaptation of the instrument with the thorax. Immediate or *direct auscultation* of the chest, the latter covered with a towel, is often a necessity in rapid examinations; but when it is necessary to analyze circumscribed sound the stethoscope is always preferable.

Gabritchewsky has recently constructed an instrument, the *pneumatoscope*, consisting of two funnels of different sizes, the smaller fitting into the larger. The former is provided with a vibrating membrane, while the latter is so constructed as to adapt itself to the patient's mouth. The apparatus is connected by two rubber tubes with the ears of the observer. The patient supports the apparatus in front of the mouth and is instructed to breathe quietly through the nose. By this means, sounds heard within the lungs and produced by percussion are said to be conveyed to the ear.

Auscultation in Health.—*Bronchial breathing* is heard over larynx, trachea and bronchi. Both inspiratory and expiratory sounds resemble the utterance of the aspirate H. The expiratory is louder and longer than the inspiratory sound. It is also likened to the sound produced by blowing down a tube of the same bore as the main bronchus. The best situation for hearing it is at the 7th cervical vertebra. It is also heard over the upper part of the sternum and the inter-scapular region at the point of bifurcation of the trachea.

The results achieved in auscultation are dependent in a measure on the way the patient breathes. The patient should be instructed how to breathe, before auscultation is attempted, and cautioned to make no noise with the mouth. If this is impossible, let the patient breathe through the nose, with closed mouth.

Origin of Bronchial Breathing. In the larynx, and is due to the formation of eddies after the air has passed the narrow glottis.

Vesicular Murmur.—This is heard over the chest where the lungs are adjacent to the thorax, excepting at points where the respiration is bronchial. It is a soft, breezy sound, heard usually only during inspiration, and is loudest at the end of inspiration. During expiration it is an uncertain murmur, and very often absent. By saying *f*, or *v*, softly, vesicular breathing may be imitated. *Puerile respiration* is heard in children, and is the vesicular breathing exaggerated.

Origin of Vesicular Murmur. In the larynx. It is bronchial breathing conducted to the ear and modified by normal lung.

The following simple experiment will demonstrate that the vesicular murmur is bronchial breathing modified by normal lung: If airless tissue, *e. g.*, a piece of liver, is placed over the larynx, and the stethoscope is placed over the liver, bronchial breathing is heard. If the same experiment is tried with a lung which has been inflated, a vesicular murmur is heard.

Intensity of Vesicular Murmur. This is dependent on force of respiration, elasticity of thorax, and density of lung tissue. It is heard more distinctly on the right than the left side, although Stokes maintains that it is louder on the left side. The vesicular murmur is most distinct where the lung is thinly covered. It is loud in the infra-clavicular, and feeble in the supra-scapular region. In $\frac{1}{4}$ of all normal chests, only inspiration is heard in ordinary respiration.

Bronchial Breathing differs from Vesicular Murmur: 1. By its higher pitch. 2. By its occurrence in inspiration and expiration. 3. By its blowing character during expiration. 4. By the distinct pause between inspiration and expiration.

The difference between bronchial breathing and vesicular murmur is with difficulty appreciated by the beginner. In cases of doubt it is best to auscultate in those situations where bronchial breathing is normally heard, and by comparison the character of the sound is easily determined.

Auscultation in Disease.—1. Changes in vesicular murmur. 2. Bronchial replaces the vesicular murmur. 3. Undetermined respiratory murmur. 4. Dry râles. 5. Moist râles. 6. Crepitant râles. 7. Pleural friction sound.

Changes in Vesicular Murmur: 1. Diminished. 2. Increased vesicular murmur. 3. Prolonged expiration. 4. Jerking respiration. 5. Systolic vesicular respiration.

Diminished vesicular murmur occurs in obstruction of the bronchi, emphysema, exudations in pleural cavity and when breathing is painful. *Increased vesicular murmur* is observed in narrowing of the bronchi, *e. g.*, in bronchial catarrh. *Prolonged expiration* is heard when the exit of air from the lung is prevented, either from loss of elasticity of the lung (*emphysema*) or obstruction of the bronchi (*bronchitis*). *In health*, the expiratory sound when heard is not one-third as long as the inspiratory sound. *Jerking respiration* is heard when inspiration instead of being continuous is interrupted; present in bronchitis. *Systolic vesicular respiration* is heard about the cardiac region, when the heart is increased in action, the contraction and dilatation of the same influencing the entrance of air into the lung.

Bronchial breathing replaces the vesicular murmur when the murmur arising in the larynx is transmitted unchanged through consolidated lung to the chest wall, *e. g.*, in pneumonia, infarction, tumors, compression of lung above pleural exudation, etc. If bronchus leading to consolidated area is obstructed (mucus, fibrin, etc) no bronchial breathing is heard, but the same appears when the offending object is dislodged by coughing. Bronchial breathing is also heard in *open pneumothorax* and over lung cavities as amphoric respiration.

Forms of bronchial breathing. Amphoric and metamorphosing respiration.

Amphoric respiration is imitated by blowing into an empty jar. It is bronchial breathing possessed of a metallic character. It is heard over large lung cavities with dense walls and in pneumothorax.

Metamorphosing respiration is characterized by an inspiration which begins with a vesicular murmur and then passes over into bronchial breathing. It is a certain sign of a cavity and is explained by the air reaching the cavity through a narrow bronchus, which, in the second part of inspiration is dilated by the air current.

Undetermined Respiration represents a form of breathing bearing the character of neither the vesicular nor bronchial.

Dry Râles are heard in catarrh of the bronchi which yield a viscid secretion with swollen mucous membrane. Air passing through the narrow tubes produces the sound. They are divided into *Sibilant* and *Sonorous Râles.* The former are whistling or hissing sounds occurring in the smaller bronchial tubes. Sonorous râles are snoring or cooing sounds produced in the larger air tubes.

Moist Râles are produced by the breaking of bubbles of air in fluid (blood, mucus or pus). They are *coarse* or *fine* according to their origin in the large or small bronchial tubes. They are modified or disappear on coughing owing to dislodgment of mucus.

They are heard during inspiration and expiration when râles are present in abundance, otherwise only during inspiration. They are common in *bronchitis* after secretion has become established. Also present in cavities and when blood and pus are present in the bronchi.

Metallic or Ringing Râles are heard added to respiratory cough or voice sound in a large cavity containing air.

Bell sound is obtained by percussing the chest with two coins, one used as a pleximeter, the other as a plessor when a ringing sound is heard. Present in *pneumothorax.*

Metallic Tinkling Râles are associated with amphoric respiration and are produced by drops of purulent matter falling from a large cavity containing air and fluid. Also heard in pneumothorax.

Crepitant Râles occur in the smallest bronchioles and air cells and are due to the inspired air forcing open the air cells agglutinated by exuded lymph. They resemble the noise produced by throwing salt on the fire or rubbing the hairs together near the ear, or, better still, by moistening thumb and index finger and separating them rapidly in front of the ear.

They are, as a rule, only heard during inspiration. They are produced in the first (*crepitatio indux*) and beginning of the 3d

stage (*crepitatio redux*) of *croupous pneumonia*, in *catarrhal pneumonia* and *œdema* of the lung.

Atelectatic Crepitation is heard over collapsed portions of lung and disappears after deep inspiration.

If we remember that the tidal or breathing volume of the air amounts to only 33 cubic inches, and the complemental air, which is the air taken into the lungs by forced breathing, which, in addition to the tidal volume, amounts to about 100 cubic inches, and knowing that the average respiratory capacity of an adult is about 225 cubic inches, the conclusion is evident, that even in a state of health the lungs are imperfectly ærated, and in a condition of *physiological atelectasis*. My invariable custom before conducting an examination of the lungs is to have the patient make repeated forced inspirations. In this way I avoid many errors in auscultation and percussion.

Sub-crepitant Râles are heard during inspiration and expiration, and are thus distinguished from crepitant rales. They are produced by air currents breaking through mucus in small bronchial tubes. Heard in *capillary bronchitis.*

Pleural Friction Sound. The respiratory rubbing of the layers of the pleura occurs in health without noise. The presence of a fibrinous exudation on their surfaces (*pleuritis sicca*), causes superficial noises (*pleuritic friction*) heard in inspiration and expiration. On palpation, pleuritic friction can often be felt. If fluid is present in pleural cavity no friction is heard or felt because the pleural layers are separated. The absence of friction is also noted when adhesion of pleural surfaces is present. Friction is heard in all forms of *pleuritis*, especially in the beginning and after disappearance of the serous exudation.

Differential Diagnosis between Râles and Friction Sounds: 1. Râles are continuous, friction sounds a series of interrupted jerks. 2. Cough changes character of râles while friction sound is not influenced. 3. Râles are diffused, friction sounds confined to a smaller space. 4. Pressure with stethoscope in pleuritis is painful, and friction sounds are increased, râles are uninfluenced. 5. Friction sounds are superficial and increased by forced inspiration. Dr. Bruen directs attention to the value of making the chest wall immovable. When the chest is fixed, especially at the lower two-thirds, by the hands of an assistant, and the stethoscope or ear is applied over the doubtful sounds, they will be found to have disappeared if of pleural origin, but to be still pres-

ent, if râles. Further, cause the patient to incline to the opposite side to that diseased, and place the hand of the diseased side on the head; this puts the pleura in a state of tension, and will often obliterate a friction râle.

AUSCULTATION OF THE VOICE.

Vocal Resonance.—During phonation, auscultation of the normal chest, except in situations where bronchial breathing is heard, distinguishes the voice as a confused hum, and words can not be recognized owing to the poor conducting quality of the normal lung.

Bronchophony.—When voice as heard over larynx, trachea or bronchi, is conducted to chest, so that spoken words become distinct. It has the same significance as bronchial respiration. As a pathological condition it always denotes consolidation of pulmonary tissue for the lung in this state is a better conductor of sound than when in a normal condition.

Pectoriloquy.—This is exaggerated bronchophony. It is heard over large and superficial lung cavities, and has an amphoric character.

Ægophony.—A variety of bronchophony. A high trembling voice and sound likened to the bleating of a goat. It is observed when the voice reaches the chest wall intermittingly. Present in incomplete compression of the bronchi, and at the upper border of a medium-sized pleuritic exudation. In speaking with the nose closed this sound may be imitated.

Metallic Sound of the Voice is heard over large cavities and in pneumothorax.

Whispered Voice.—The vibrations produced by this voice in the normal condition of the lung tissue are too feeble to be appreciated. When solidification of the lung or a cavity exists the whispered voice is transmitted with unusual distinctness (*whispering bronchophony*).

Bacelli observed that in serous exudations of the pleura, the whispered voice was conducted to the ear; whereas, when the

exudation was purulent, it was not. This sign, called the *Phenomenon of Bacelli*, was supposed to be of importance in differentiating serous from purulent exudations; but, not being always present in serous, and being occasionally heard in purulent exudations, it loses much of its practical importance.

SUCCUSSION.

This is a splashing sound, produced when air and fluid are present in the pleural cavity at the same time (*sero-* or *pyo-pneumothorax*). The sound is somewhat similar to that obtained by shaking a cask containing air and fluid. The direction of *Hippocrates*, in order to obtain this sound, was to shake the patient by grasping the shoulders, and with the ear applied to the chest it could be heard.

The succussion sound is sometimes so loud that it can be heard in every part of a room. The less fluid and the greater quantity of air, the louder the splashing. Being most pronounced in *pneumo-hydrothorax*, it may be heard to a lesser degree over very large cavities with fluid contents.

Examination of the Thymus Gland. This ductless gland is a temporary organ, attaining its full size about the age of two years. It subsequently atrophies, until at puberty it has almost disappeared. It consists of two lateral lobes, and extends from the 4th costal cartilage to the lower border of the thyroid gland. In the mediastinum it rests upon the pericardium. In children, *percussion* will indicate the presence of this gland by a finger breadth dullness in the left sternal line, extending from the 2d to the 4th costal cartilage. The thymus gland may be hypertrophied, or the seat of hæmorrhage, abscess, tuberculosis, tumors, etc. Dullness at the upper part of sternum may also be due to aneurism of the arch of aorta, mediastinal tumor, or a substernal struma.

Puncture of the Pleura is practiced with an ordinary hypodermic syringe, with antiseptic precautions. Its object is to determine the presence and character of fluid in the pleural cavity. It is a harmless procedure, and is further indicated in diagnosing pleuritis from other affections (*pneumonia, tumors, thickening of pleura, etc.*) The character of the exudation in pleuritis may be serous, sero-fibrinous, sero-purulent, purulent, or hæmorrhagic.

Microscopical Examination of Fluid may reveal the presence of streptococci, actinomyces, and bacilli of tuberculosis, although the absence of the latter does not exclude tuberculous pleuritis. Carcinomatous cells may be present in carcinomatous pleuritis.

ASSOCIATION OF THE PHYSICAL SIGNS OF THE LUNGS.—*(Altered from Da Costa.)*

PERCUSSION	AUSCULTATION OF RESPIRATION.	AUSCULTATION OF VOICE.	VOCAL FREMITUS.	PHYSICAL CONDITION.
Clear	Vesicular murmur or its modifications	Normal vocal resonance	Unimpaired	Normal (*or nearly*) lung tissue
Dull	Bronchial respiration	Bronchophony	Increased	Consolidation of the lung
Dull	Absent respiration.	Absent voice	Diminished or absent	Effusion into pleural cavity; obstruction of the bronchus associated with consolidation of the lung.
Tympanitic	Cavernous, or feeble, according to cause.	Cavernous or diminished	Increased, diminished or absent	Cavities of the lung; relaxation of the lung tissue; pneumothorax.
Amphoric or metallic	Amphoric or metallic.	Amphoric or metallic	Increased or diminished	Large cavity with elastic walls; pneumothorax.
Cracked metal sound	Cavernous respiration.	Cavernous voice.	Uncertain, increased or diminished	Generally a cavity of the lung communicating with a bronchial tube

PHYSICAL DIAGNOSIS OF RESPIRATORY DISEASES

DISEASE.	INSPECTION.	PALPATION.	MENSURATION.	PERCUSSION.	AUSCULTATION.
BRONCHITIS.	In *capillary bronchitis*, inspiratory retraction of thorax.	Bronchial fremitus. Vocal fremitus normal.	Normal.	Normal.	Dry râles when fluid is viscid. Sonorous in large, and sibilant râles in small tubes. Prolonged expiration. *Mucous râles* when secretion is free.
BRONCHOSTENOSIS.	Thoracic movements on affected side diminished. Inspiratory dyspnœa and inspiratory retraction of chest.	Vocal fremitus on affected side diminished.	Circumference of thorax on affected side diminished.	Normal in beginning but soon followed by a tympanitic sound (*sign of lung relaxation.*)	Vesicular respiration diminished or absent. Whistling or hissing sounds.
BRONCHIECTASIS.	Flattening of affected side due to retraction of connective tissue around dilated bronchi. Inspiratory retraction of chest.	Large bronchiectases when superficial and surrounded by consolidated lung cause increased vocal fremitus.	Normal or diminished circumference.	When dilatation is superficial and large, tympanitic or tympanitic metallic sound. Other signs of cavities. Signs absent when cavities are filled with secretion but reappear when contents are expectorated.	Bronchial respiration with metallic sounds when cavities are superficial, otherwise vesicular respiration. Moist râles when fluid is present in cavities.
BRONCHIAL ASTHMA.	Expiratory dyspnœa. Chest semi-distended with little movement.	Bronchial fremitus.	Circumference of thorax increased during paroxysm.	Hyper-resonance. Immobility of lung borders. Absolute cardiac dullness diminished owing to acute inflation of lungs.	Diminished vesicular respiration and sibilant râles heard loudest during expiration. They become moist at termination of attack

PHYSICAL DIAGNOSIS OF RESPIRATORY DISEASES.—*Continued.*

DISEASE.	INSPECTION.	PALPATION.	MENSURATION.	PERCUSSION.	AUSCULTATION.
ATELECTASIS.	Inspiratory retraction of chest especially at its lower part. Superficial and rapid respirations.	Increased vocal fremitus when dullness is pronounced.	Circumference of affected side diminished.	Weak percussion gives dullness when airless lung is at least 2 cm. thick and 5 cm. wide and superficial.	Bronchial respiration and bronchophony when dullness is pronounced.
EMPHYSEMA.	Barrel-shaped chest, and no appreciable movement on inspiration. Expiratory dyspnœa. Supra-clavicular regions bulged on coughing.	Thoracic movements diminished. Vocal fremitus diminished owing to diminished thoracic vibration.	All diameters increased.	Hyper-resonance. Liver and heart dullness diminished. Immobility of lung border during respiration.	Respiratory sounds diminished. Prolonged expiration. Sibilant and sonorous râles.
ŒDEMA OF LUNG.	.....	.....	.....	When amount of fluid is enough to replace air (*rare*) dullness. Normal when fluid is not excessive; if altered it becomes tympanitic.	Bronchial respiration and bronchophony when fluid is sufficient to supplant air. Crepitant and bubbling râles.
CROUPOUS PNEUMONIA.	Affected side shows little movement. Respirations increased. Pulse and respiration ratio changed from 4 to 1 to 2 or 3 to 1.	Vocal fremitus increased; this is not the case when main bronchus is obstructed or when extensive infiltrations make thorax too tense.	Affected side shows increased measurement.	Tympanitic sound in first stage and resolution. When infiltration is complete dull percussion note, but not flat as in pleuritic exudations.	Crepitant râles in beginning of infiltration and in stage of resolution, and occurring as a rule only during inspiration. When infiltration is complete, bronchial respiration.
CATARRHAL PNEUMONIA.	Dyspnœa. Inspiratory retraction of chest.	Increased vocal fremitus when large lung districts have been made airless.	.....	Dullness when extensive infiltration. Dullness usually bilateral and not confined to lobes of lung but extends alongside vertebral column.	Usual auscultatory signs of dullness when infiltration is extensive enough; otherwise moist râles.

PHYSICAL DIAGNOSIS OF RESPIRATORY DISEASES.—*Concluded.*

DISEASE.	INSPECTION.	PALPATION.	MENSURATION.	PERCUSSION.	AUSCULTATION.
PHTHISIS PULMONALIS. *	Paralytic thorax. Frequent and shallow breathing.	Feeble expansion of chest. Vocal fremitus increased in affected portions in all stages.	Reduced circumference and expansion.	Dullness over affected districts. Tympanitic sound over cavities and cracked-pot sound if cavity communicates with a bronchus.	Harsh, jerky or prolonged expiration. Crepitant râles over apices in early stages. Bronchial respiration and bronchophony in consolidation. Gurgling râles and cavernous or amphoric breathing over cavities.
PLEURITIS SICCA.	Patient lies on unaffected side. Retarded respiratory movements.	Friction fremitus. Pain on pressure in intercostal spaces. Vocal fremitus not altered.	Not altered.	Normal.	Pleuritic friction sounds, increased in intensity by pressure with stethoscope, and heard during inspiration or expiration, or both.
PLEURITIS HUMIDA.	Patient lies on affected side. Respirtory movements diminished or absent. Intercostal spaces bulged. Displacement of apex beat.	Vocal fremitus impaired or absent. Fluctuation rarely felt. Dislocated organs can be palpated	Increased circumference of affected side which is often very pronounced.	Absolute dullness altering on change of position. When fluid is small in quantity dullness is first detected in posterior lower part of chest.	Vesicular respiration diminished or absent. Bronchial respiration, when lung has been compressed. Vocal resonance diminished or absent. Pleuritic friction sounds above exudation and at time of resorption.
PNEUMOTHORAX AND HYDRO PNEUMOTHORAX.	Affected side bulged with obliteration of intercostal spaces. Apex beat dislocated. Movements of affected side diminished.	Vocal fremitus diminished or absent. When fluid is present, splashing sound on shaking patient.	Affected side shows increased measurement.	Over fluid, dullness; above same clear or tympanitic sound. Heart's dullness dislocated toward sound side.	Succussion sound, and metallic tinkling. Absence of vesicular breathing. Amphoric respiration when perforation is not closed.

* Prof. KOCH claims to have discovered a fluid by the aid of which doubtful cases of phthisis may be diagnosed. The same is likewise true of affections of the glands, latent tuberculosis of bone, doubtful cases of skin tuberculosis, etc. A dose of 0.01 cubic centimetre of this fluid injected subcutaneously into tuberculous patients is followed by an attack of fever, beginning with rigors, and the temperature rises above 39° C; there is also vomiting, pain in the limbs, coughing and great fatigue. The attack begins 4 or 5 hours after the injection and lasts from 12 to 15 hours. The healthy human being reacts either not at all or scarcely at all when 0.01 cubic centimetre is used.

THE PHONOGRAPH IN MEDICINE.*

A few years have elapsed since Edison devised an instrument consisting essentially of a mounted diaphragm, so fixed as to operate a stylus which recorded the inflections of the voice on tin foil. With this crude apparatus, which sacrificed distinctness of articulation in order to secure a loud tone, not only was sound recorded, but reproduced. Attention has of late again been directed to an improved phonograph, embodying the same principles as the original instrument, but so modified as to be of practical value. It is now but a few months since the untiring Edison, dissatisfied with his previous achievements, added further improvements to the phonograph, making it now automatically adjustable and combining the recorder and reproducer in one. The perfected phonograph requires no dexterity in manipulation, and I will briefly describe the method of taking a phonogram with it.

To record sound, a wax cylinder is fitted on to a metal one. The diaphragm, which is automatically adjustable, is then made to rest on the wax and the instrument is ready for use; on speaking into a tube, which is in communication with the diaphragm, the sound pulsations are incised by means of a small point in the noiselessly revolving cylinders. Upon examining the wax cylinder, a number of fine lines can be seen, which are an absolute equivalent for the sounds made by the voice. All that is necessary to reproduce the sounds is to move a lever, when another self-adjustable point is presented, which passes over the indentations in the wax made by the recording needle, and absolute reproduction of the voice, even to the lightest shades and variations, results. This may be repeated an indefinite number of times. The wax cylinders cost twenty-five cents apiece, making phonograms for permanent record very expensive. This objection will soon be obviated, as mailable cylinders, costing but a few cents and available for single records, are to be introduced.

To run the machine, either an electric motor or foot power may be used. I use the latter, as the instrument is under better control: An electric motor, however, possesses the advantage of rendering the revolutions of the wax cylinder almost noiseless an indispensible factor in the reproduction of fine sounds. The accurate reproduction of the human voice, is well illustrated by the following occurrence: A lady on hearing the words of a gentleman, spoken into my instrument but a few days before, at once exclaimed: "I know that voice! It is the voice of Mr. H———." I asked her if she knew the gentleman and she replied in the neg-

*Reprint from *Occidental Medical Times*, April, 1890.

ative, but she had heard him recite once at an entertainment many months before. Here is an instance of how unerring is the memory for sounds.

I have employed the phonograph in taking the histories of cases, thus saving time and obtaining copious notes. The cylinders can be transcribed at the convenience of the amanuensis. This is but one of the many practical uses to which it may be put. In physical diagnosis the application of the phonograph suggests itself, and it is claimed, that in the laboratory of Edison, the normal heart sounds were first successfully recorded. Its practical value in medicine was tested by the Paris Academy of Sciences and the Vienna Medical Society, with results largely in favor of the instrument. Dr. Bleyer, in an article on "The Phonograph in Physical Diagnosis," says that by means of a microphonic stethoscope, he was enabled to record all the thoracic sounds in their varied pitch and tone. The sounds thus obtained were projected by means of a tin cylinder acting as a resonator and directly instructed a large class of students. I must confess, that similar experiments faithfully executed with all due regard to details, were unattended by these results. I found in my investigations, which were varied in every possible manner, that the recording of heart sounds was practically impossible. The fault rests with the phonograph. There is no question about the sensitiveness of the diaphragm for recording even the feeblest sounds, but the difficulty lies in reproduction. Even loud sounds emanating from the chest are with difficulty detected by an ear accustomed to the phonograph. When the reproducing needle is adjusted to the revolving wax cylinder a hissing sound is heard, the result of friction between the needle and the wax. It is this sound which interferes with proper reproduction, and I have found that lubricating the cylinder renders reproduction decidedly more distinct. If this objection were obviated, the phonograph would prove an ideal instrument in physical diagnosis.

Many sounds heretofore inappreciable can be brought out by the phonograph. The deepest tone that we are able to appreciate contains 16 vibrations a second; the phonograph will record 10 vibrations or less, and can then raise the pitch until we hear a reproduction from them. Similarly, vibrations above the highest rate audible to the ear can be recorded and then reproduced by lowering the pitch. I have been able to record nearly all the percussion sounds of sufficient intensity, and with a degree of distinctness which is remarkable. The percussion sounds obtained in this way are exaggerated, and the method resembles auscultatory percussion. The fine lines which can be seen on the wax cylinder after talking into the phonograph convey a fair idea of the number and variety of air waves necessary to the production of words and sentences.

In clinical medicine the uses of the phonograph are varied. The different anomalies of speech, whether due to paresis, paralysis, tremor, spasm, or even ataxia of the muscles of articulation,

can be faithfully recorded. Coughs of various kinds can also receive accurate registration.

Determining the sense of hearing is of great importance to the aurist, and the present apparatus for this purpose is unsatisfactory. Roosa, in his text-book, refers to the characteristic answer of a boy who, upon being tested by the ticking of a watch, suddenly exclaimed: "What do I care about hearing a watch? I want to hear what people say!" Dr. Lichtwitz, in a recent article on "The Phonograph in Otology and Laryngology," considers the following as essential to an apparatus for ascertaining the perception of sound; (1) It should produce all the tones and murmurs appreciated by a normal ear, and especially speech in all its inflections. (2) It should be a constant sound producer. (3) It should be an universal apparatus, so that otologists in different countries can express themselves definitely regarding the acuteness of hearing, as ophthalmologists do in testing vision. (4) Its use should be unaccompanied by inconvenience. (5) It should allow of the perception of sound by bone and air conduction. The phonograph, according to this author, absolutely subserves the first two indications, and is the best apparatus for determining the sense of hearing.

Of all the wonderful accomplishments possessed by the phonograph, its availability for the blind, by placing them in communication with the world of literature, far exceeds any similar device for the relief of this unfortunate class. By the majority of those whose eyesight is impaired or destroyed it will be hailed as a veritable God-send. Each wax cylinder will receive from 800 to 1,000 words, and the whole of "Nicholas Nickleby" can be put on four cylinders eight inches long with a diameter of five. By a mechanical process these cylinders can be multiple-copied, which will render them comparatively inexpensive. For educational purposes, as in the study of foreign languages, which to physicians is often a necessity, the phonograph is beyond comparison, for no system of phonetic spelling or verbal description will convey the same pronunciation of a foreign language so well as a machine that reproduces the human voice.

The phonograph is not without a therapeutic use. The psychic influence of sound cannot be denied, and we need only recall the well-known effects of enlivening music in cheering troops on the march or while under the depressing influence of siege, and the profound influence of certain music in exciting the highest emotional centres. When music, and good music at that, can be had "on tap," and supplemented by stories, songs and recitations by well-known authors, vocalists and elocutionists, the convalescent may command a variety of entertainment.

CHAPTER V.

COUGH—SPUTUM.

Cough is a symptom of disease of the respiratory apparatus. It is a reflex act, the result of *laryngeal, tracheal,* or *bronchial irritation.* The chief object of this act is the expulsion of pathological products which, if allowed to accumulate, would result primarily in dyspnœa, to be followed by asphyxia. For this reason, the use of *narcotics* which render anæsthetic the respiratory mucous membrane, are dangerous when the secretions are abundant. The probable direct cause of cough is irritation of the fibres of the *pneumogastric* nerve, or its branches. A *cough centre* is supposed to exist in the floor of the 4th ventricle. Cough may result from irritation of the *pharynx, œsophagus, stomach, liver, spleen,* and *female genitalia.* In many people, cough may be produced by touching the nasal mucous membrane with a sound, and many intractable spasmodic coughs have been cured by the removal of nasal polypi. In about 17 per cent. of persons, cough will follow *irritation of the external auditory meatus.* In *hysteria* a persistent cough may be present. We also speak of a *nervous cough.* Irritation of the alveoli of the lung will not result in cough, and secretions must first reach the communicating bronchus, before coughing is produced. Kinds of cough: 1. *Dry cough* is present in pleuritis, pneumonia (1st stage), acute bronchitis, and broncho-pneumonia. 2. *Moist cough,* when the air passages are filled with an abundant secretion. 3. *Brassy* (barking) *cough* is heard in stenosis of the larynx (croup). This cough is usually associated with *stridulous respiration,* when pressure is exerted on the trachea (*Aneurisms, tumors*). 4. *Whooping-cough* in pertussis. It is a long-drawn, crowing sound, coincident with inspiration, and follows a violent series of coughs. It is due to the passage of air through the spasmodically closed glottis.

In *Laryngismus Stridulus,* a crowing inspiration resembling that of pertussis, although not usually associated with cough, is heard, and is also due to *spasm of the glottis.* A *pharyngeal cough* is frequently attended with vomiting. An *empyema* perforating the air passages, is followed by sudden and copious expectoration. In *phthisis,* cough is often dependent on the ingestion of food, and is caused by *epiglottis,* the seat of tuberculous infiltration, imperfectly closing the opening of larynx.

Signs Obtained by Coughing (*Tussive signs*).—*Auscultatory signs* obtained by respiration and the voice, may likewise be obtained by acts of coughing. Coughing will remove accumulations of mucus within bronchial tubes, and thus restore a diminished or suppressed murmur over some part of chest. In coughing, more air is expelled than by an ordinary expiration, and in the following inspiration the vesicles capable of greater expansion, give rise to a proportionately loud inspiratory sound, thus intensifying normal and abnormal inspiratory sounds. *Cavernous gurgling* may be obtained very distinctly with cough. Cough changes the character of râles, leaving friction sound uninfluenced.

SPUTUM.

Origin of the Sputum.—The constituents are furnished by the secretions of the pulmonary alveoli, bronchial tubes, larynx, pharynx, mouth and the nares with their adjacent cavities. In health the secretion from the air-passages does not exceed in quantity that required to moisten the mucous surfaces. In disease the secretion may be in excess and contain many new constituents. Children and aged individuals usually swallow the sputum. *Chemical analysis* of the sputum is rarely of diagnostic value. In the examination of the sputum we note: 1. *Consistency.* 2. *Color.* 3. *Odor.* 4. *Quantity.* 5. *Reaction.*

Consistency is dependent on the quantity of mucus. In *pneumonia* the sputum is very viscid while purulent sputum has little consistency.

Color.—*Whitish* or *colorless* in mucous, *yellowish* in muco-purulent, and *greenish* in purulent sputum.

When the *blood-coloring matter* in the sputum is altered the color imparted may be red, brown or yellowish-green. *Yellow-ochre* color (*presence of hæmatoidin*) is present in abscess of the lung. A *green color* (bile pigments) is met with in pneumonia complicated by icterus. A green color may also be caused by micro-organisms. The sputum may be colored *blue* (laborers in dye works), *black* (coal laborers) and *ochre-colored* and *red* (iron-workers.)

Odor.—Very fetid in dilated bronchi, putrid bronchitis and gangrene of the lung.

Quantity.—Daily estimation is of value in controlling the progress of a case. The quantity is large in purulent

bronchitis, dilated bronchi, tuberculous cavities, œdema, abscess, etc.

Reaction.—Usually alkaline.

According to the principal constituents, the sputum may be divided as follows: 1. *Mucous.* It resembles the natural secretion. It is tough, transparent and colorless, and is present in beginning bronchitis. 2. *Purulent.* It is of a greenish-yellow color, and is found in rupture of abscesses from the lung or neighboring organs, empyema, chronic bronchitis (*bronchorrhœa*), etc. 3. *Serous.* Pathognomonic of lung-œdema. 4. *Bloody* (*hæmoptysis*). Present in the first stage of tuberculosis (*initial hæmoptysis*) or in any part of its course: in cardiac disease when venous stasis develops (particularly in mitral lesions): aneurisms (bleeding profuse); hæmorrhagic infarctions, etc.

We also distinguish the following mixed forms: 1. *Muco-purulent* in bronchitis (mucus and pus intimately mixed) and phthisis. In the latter affection, the mucus and pus are not mixed and the pus is in the form of balls covered with mucus which sink in water (*sputum globosum*).

2. *Sanguineo-mucous* found in carcinoma of the lung (*raspberry-jelly sputum*), pneumonia (1st. stage), and hæmorrhagic infarction.

3. *Sanguineo-serous* (*prune-juice sputum*) is found in œdema of the lung and pneumonia. Blood-colored saliva may be expectorated by hysterical individuals who often prick and suck their gums: the blood is more watery than usual and is generally not ærated. *Shreds of the parenchyma of the lung* may be found in abscess and gangrene of the lung.

With the microscope the following constituents may be found in the sputum:

1. *White Blood Corpuscles.* Always present, and especially abundant in purulent sputum.

2. *Red Blood Corpuscles.* Present in bloody sputum. Corpuscles are preserved in form, although pale.

3. *Epithelium. Pavement* (Fig. 8, a) from the buccal cavity and true vocal cords. *Cylindrical*, from the nasal cavity, upper part of pharynx, the larynx and bronchi. *Alveolar* (fig. 8, b) from the alveoli of lungs and finest bronchi. They are large, round or oval cells, and many contain fat, carbon and myeline. They are of no special diagnostic importance, but their presence in large quantities might suggest tuberculosis.

4. *Crystals. Charcot-Leyden Crystals* (Fig. 8, c). Colorless pointed octahedra, insoluble in cold water, ether, alcohol or chloroform, but easily soluble in alkalies, mineral acids, warm water and acetic acid.

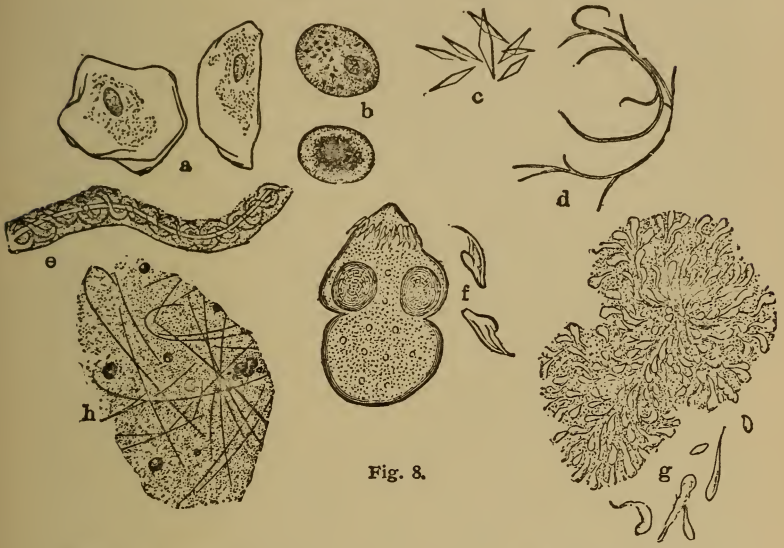


Fig. 8.

Fig. 8. a. Epithelium from mouth. b. Epithelium from lung alveoli containing granules of black pigment. c. Charcot-Leyden crystals. d. Elastic fibres. e. Spiral of Curschmann. f. Echinococcus (scolex and hooklets). g. Actinomyces. h. Needles of margaric acid.

They may at times be seen with unaided eye in yellowish masses found in the sputum. They are met with in *bronchial asthma* (diagnostic) during and after the paroxysm. Also observed (rarely) in acute bronchitis and tuberculosis.

Hæmatoidin Crystals appear as amorphous yellow-brownish grains, or in rhombic plates and needle-like crystals. They come from extravasated blood which has been retained for some time in the air passages.

Needles of Margaric Acid (Fig. 8, h). Soluble in ether or chloroform. Found in gangrene and putrid bronchitis inclosed in minute plugs which emit an intensely foul odor.

Cholesterin Crystals. Rhombic plates (TEST: *with diluted sulphuric acid and tincture of iodine, a green then a red color*). Found in old purulent sputum. Crystals of leucin and tyrosin are often seen in conjunction with cholesterin.

Crystals of Oxalate of Lime (in diabetes) and *triple phosphate* (when sputum has undergone decomposition with formation of ammonia) have also been observed.

5. *Spirals of Curschmann* (Fig. 8, e). May be recognized with unaided eye as fine threads in small sago-like clumps of mucus. They are spiral-shaped bodies with a bright central fibre. Curschmann supposed that they were diagnostic of bronchial asthma, but they are also found in capillary bronchitis, croupous pneumonia and pulmonary tuberculosis. They probably consist of mucin.

6. *Corpora Amylacea.* Look like amyloid grains, and are without clinical importance.

7. **Entozoa.**—*Echinococcus hydatids* (Fig. 8, f). May develop in the lung or come from the liver; *Ascaris lumbricoides* derived by migration from intestinal tract; Eggs of *Bilharzia hæmatobia.*

8. **Bronchial Fibrinous Casts.**—They are moulds of the finer bronchi, and occur in fibrinous inflammations (croupous pneumonia, fibrinous bronchitis).

9. *Elastic Fibres.*— (Fig. 8, d.) Observed in all destructive affections of the lung (tuberculosis, gangrene, abscess), and rarely from the mucous membrane of the respiratory tract, vocal cords or epiglottis. In certain cases of lung gangrene the elastic fibres are absent, owing to the formation of a ferment which dissolves them. They are recognized by their transparency, sharpness of outline and noose-shaped arrangement. They stain with a watery solution of magenta.

Elastic fibres are also derived from the food, hence the necessity of washing the mouth before the sputa are collected. In order to find the elastic fibres, select a part of the sputum (caseous plugs preferred), and mix it on a slide with a drop of a ten per cent. solution of *caustic potash.* When the elastic fibres are not abundant, mix all the sputa collected in 24 hours with an equal bulk of caustic potash, and boil until the mass is fluid. (If boiled too long, elastic fibres lose their characteristic appearance); now pour the liquefied sputum into a conical-shaped glass, and examine sediment for the fibres (*Fenwick's method*). Elastic fibres should only be diagnosed when they show an alveolar arrangement, or when at least several adhere together. The number and mass of elastic fibres convey some idea of the extent and rapidity of the destructive process.

10. **Micro-Organisms**, *Leptothrix buccalis, sarcinæ, tubercle-bacillus, pneumonia microbes, micro-cocci and bacilli, actinomyces*, and *aspergillus*. *Leptothrix threads*, when found in the mouth, are without diagnostic significance. They are present in the bronchial plugs in putrid bronchitis. They are stained blue by a solution of *iodine* in *iodide of potash*. When this reaction is not practiced, they may be confounded with elastic fibres or fatty acid crystals.

Sarcinæ pulmonis rarely occur in the sputum, and are without clinical importance.

Tubercle Bacillus (*Koch*).—Found in the sputa in pulmonary and laryngeal tuberculosis. Likewise in tuberculosis of the nasal or pharyngeal mucous membrane (rare). The presence in the sputum of the bacilli may precede all other physical signs of pulmonary tuberculosis; and the diagnosis of the latter affection is only then certain when the specific organisms have been demonstrated. (See page 56.) A failure to find the bacillus, after one examination, does not exclude tuberculosis; on the contrary, many examinations are often necessary. The bacilli are few in number in the incipient stages of tuberculosis, and their presence in the sputum may be intermittent in all stages of the disease, when the bronchus communicating with the seat of bacillary destruction has been occluded, or when a temporary arrest of the destructive process has occurred with retention of the bacilli in the tissues. Errors in "*technique*" must also be taken into consideration, and when in doubt about the reliability of staining solutions, etc., a specimen known to contain the bacilli must be stained at the same time. An Abbe substage condenser is a decided aid to the discovery of the bacilli in cover-glass preparations; but when this is not obtainable, flood the specimen with as much light as possible. The microscope should magnify at least 450 diameters.

Methods of Staining for the Tubercle Bacillus. *Method of Ehrlich.*—The following solutions are necessary: Solution (1). Aniline water (a filtered saturated solution of aniline oil in water). Solution (2). Concentrated alcoholic solution of fuchsin. Solution (3). Diluted nitric or hydrochloric acid (1:3 water). Solution (4). Concentrated watery solution of methyl blue.

Selection of sputum and preparation of cover glass.—Pour the sputum in a vessel with a black back-ground (a black vulcanite dish or porcelain plate painted black will do). With a needle previously sterilized by heating in the flame of a lamp, search for

minute opaque white points about the size of a pin's head or yellowish masses; if both are absent select from the purulent portion. Now place a very small quantity of the selected material between two cover glasses, which are rubbed together until a thin film of the matter is deposited on each. The separated covered glasses, protected from dust, are now allowed to dry. When dry pass cover glasses three times quickly through a spirit flame to fix the preparation and coagulate the albumen, when they are ready for staining.

Staining.—To solution (1) in a watch glass add enough of solution (2) until the fluid becomes turbid (about six drops are necessary). Now float cover glasses in this solution and allow them to remain protected from the dust for 24 hours. If time is an object staining may be hastened by first heating the solution, as prepared above, in a test tube and then pouring it into a watch glass to which is then added the glasses, which remain in the solution for about ten minutes. The cover glasses are next dipped for a few seconds into solution (3) and then washed in water. In this solution the glasses must not remain too long, or the bacilli will be decolorized. To prevent this possibility a concentrated solution of oxalic acid has been recommended, but I do not find this satisfactory. After removal from solution (3), all micro-organisms are decolorized except the tubercle-bacillus (and *lepra-bacillus*). To color the remaining portion of the specimen (the tubercle-bacilli only being colored), a drop or more of solution (4) is added to the cover glass and allowed to remain in contact for about one minute. Now wash in water thoroughly and dry the cover glass, if it is to be examined in cedar oil or Canada balsam. The bacilli of tuberculosis, if present, will be seen as intensely stained red rods on a blue back-ground. A watery solution of malachite-green may be substituted for solution (4), the green contrast stain making the red bacilli very evident.

For rapid staining with ready prepared solutions, the following method of Gabbet will give excellent results:

Solution (1)	Fuchsin,	1.0
	Absolute alcohol,	10.0
	Carbolic acid,	5.0
	Distilled water,	100.0
Solution (2)	Methyl blue,	2.0
	Sulphuric acid,	25.0
	Distilled water,	100.0

The cover-glass preparation is placed in solution (1) for 10 minutes, then washed in water and immersed for 3 minutes in solution (2); washed again in water, and examined. The bacilli are red, back-ground, blue.

When the bacilli are suspected to be few in number, the method of *Biedert* may be employed: Mix one tablespoonful of sputum with two of water, and add 15 drops of caustic soda; cook until entire mass is liquified, when it is put into a conical glass and allowed to remain for two days, but no longer. The sediment will contain elastic fibres and tubercle-bacilli. Stain sediment for the latter in the usual way.

Pneumo-Coccus.—The diagnostic value of this microbe is not definitely established. It is invariably present in the sputum of croupous pneumonia, although bodies practically indistinguishable from it by the aid of the microscope are found in broncho-pneumonia, and even in healthy persons. The most common variety of the pneumo-cocci consists of small, round bodies, arranged in pairs (*diplococci*), surrounded by a gelatinous capsule; they may occur singly or in strings.

The *pneumo-cocci* are best stained by *Friedlander's* method. The cover-glass preparation is put for a few minutes in a one per cent. solution of acetic acid; the excess of acid is blown off by means of a pipette, and the glass then allowed to dry. Now introduce the glass into a solution of methyl-aniline violet (aniline water, 100 parts, alcoholic solution methyl-violet, 11 parts, absolute alcohol, 10 parts), for a few seconds; wash in water; dry and mount. By this method the capsule is clearly demonstrated.

Micrococci and bacilli are found in every sputum, but are increased in fetid bronchitis, bronchiectasis, and gangrene of the lung.

Actinomyces (*ray fungus*).—Found in actinomycosis of the lung. In obscure cases simulating empyema or localized abscess in the thorax, the pus is characteristic. It contains little yellow masses as large as a millet-seed, or smaller; which show under the microscope, after being gently crushed, a large number of fine, radially arranged filaments, which end in thick knobs.

Staining of the ray fungus is not usually necessary, but when employed, picro-carmine is excellent, the actinomyces are stained yellow, the remaining parts of the specimen, red.

Aspergillus (*glaucus and niger*)—Found in the sputum of phthisis and other destructive processes of the lung. They have been identified with a condition of the lung known as *Pneumonomycosis aspergillina*. They are branched filaments without double contour and contain pigmented spores.

THE SPUTUM IN DISEASES OF THE RESPIRATORY APPARATUS.

Croupous Pneumonia.—In the early stages the sputum is colorless and small in quantity. Later, at times even a few hours after the initial chill, it becomes *rusty* and unusually viscid. In the second stage of the disease, the rusty color (due to intimate mixture of blood and sputum) is most pronounced. As the pneumonia approaches resolution, the sputum is of a citron-yellow color. If œdema of the lung complicates the affection, the sputum is increased in quan-

tity, and is fluid and foamy. In unfavorable cases, sputum looks like *prune juice*. *Green color* of sputum, when icterus complicates the pneumonia or when resolution is protracted, or when abscess of the lung follows. *Fine fibrinous bronchial casts* (detected by shaking sputum in water) belong to the stage of hepatization, appearing on the third and disappearing on the seventh day of disease. *Pneumo-cocci.*

Abscess of the Lung.—Expectoration abundant and resembles pus. Odor like butter-milk; becomes fetid when gangrene is present. Sputum in standing separates into an upper watery and lower layer consisting of pus corpuscles. Minute pieces of lung tissue may be present. *Microscopical examination:* Lung tissue and elastic fibres with alveolar arrangement.

Gangrene of the Lung.—Abundant expectoration of a fetid odor and dirty green color. Sputum separates on standing into three layers. Upper layer frothy and turbid; middle, watery and lower layer viscid and made up of a sediment of pus corpuscles, bronchial plugs containing leptothrix pulmonalis, cercomonas, fat drops, etc. Elastic fibres usually absent owing to the action of a ferment which destroys them.

Hæmorrhagic Infarction.—When recent, compact blood masses intimately mixed with mucus and of a light red color are expectorated. After several days sputum becomes brownish.

Tuberculosis.—In *acute miliary tuberculosis* no tubercle-bacilli are found. Character of sputum in *pulmonary tuberculosis* varies according to the anatomical condition existing and stage of disease. The presence of the *tubercle-bacillus* is alone diagnostic. The number of bacilli present is usually no index of the severity of the disease. They are more abundant in pyrexial than apyrexial periods. When hæmoptysis occurs, they are apparently diminished owing to dilution of sputum with blood. Elastic fibres also present, but show only lung destruction, the character of which is determined by demonstrating the tubercle-bacilli.

Pneumonoconiosis.—A condition of the lung traceable to the constant inhalation of *dust* or irritating particles. In *anthracosis*, sputum contains particles of *carbon*, and the color is gray or black. In *siderosis pulmonum* due to inhalation of *iron dust*, the sputum is of a brownish black color. Leucocytes and alveolar epithelium as in former condition are filled with pigment.

Bronchitis.—Character varies according to the form and stage of the affection. In the early stages it is mucous (*sputum crudum*), while later it is muco-purulent (*sputum coctum*). When this affection is of long standing, it may be wholly purulent. In *fibrinous bronchitis* regularly formed casts of the tubes are found. In *putrid bronchitis* odor of sputum is like that of gangrene, but it contains no elastic fibres or particles of exfoliated lung.

Bronchiectasis.—Sputum abundant, especially in the morning. Owing to stagnation it is often fetid.

Bronchial Asthma.—Sputum is muco-purulent and somewhat frothy. Asthma crystals. Spirals of Curschmann.

CHAPTER VI.

EXAMINATION OF THE HEART.

Methods: 1. Inspection. 2. Palpation. 3. Percussion. 4. Auscultation.

INSPECTION AND PALPATION.

Inspection of the cardiac region in a healthy person shows no difference in comparison with a corresponding region on the right side. A protrusion of the cardiac region is called *voussure*, which is present in enlargement of the heart and when air or fluid is present in the pericardial sac. The degree of protrusion is dependent on the size of heart, the quantity of air or fluid and the resistance of the thorax.

The heart's action is discernible in healthy persons either as a diffuse vibration of the cardiac region or as a circumscribed elevation in the lower portion of the same region. The first is *the impulse of the heart*, the second, the *apex beat*. The apex beat may be brought into prominence for clinical purposes by directing the patient to breathe rapidly or to make physical exertion of some kind.

In investigating the apex beat we observe the following: 1. Location. 2. Breadth. 3. Force. 4. Time. 5. Rythm. *The location of apex beat* is in the 5th left intercostal space between the left mammary and parasternal line. The normal location of apex beat excludes as a rule; hypertrophy, dilatation, pericardial effusion and dislocation of the heart.

Physiological change in the location of apex beat: 1. In children up to the 10th year, it may be found in 4th intercostal space and more outward. 2. In old age, it may be found in 6th intercostal space. 3. During deep inspiration it may descend one intercostal space. 4. *Position:*

Lying on the left side may dislocate apex beat to the left axillary line. Lying on the right side will not dislocate it more than 1 inch. 5. After physical or mental exertion it is diffused. 6. It is higher in short than long chests.

Causes of apex beat: 1. Change in the form of the heart during systole; its anterior-posterior diameter is increased (*Ludwig*) and apex is dislocated forward, upward, and to the right. 2. Change in the position of heart; it descends and turns on its long axis (*Skoda*).

Pathological change in location: 1. Dislocation of the heart. 2. Enlargement of the heart.

Dislocation of heart occurs in deformities of the thorax, emphysema, air and fluid in pleural sac and contraction of the lung. It is also dislocated upward, when the diaphragm is pressed upon by abdominal tumors, ascites, etc.

Enlargement of heart occurs in hypertrophy and dilatation of the left ventricle which dislocate apex beat outward or outward and downward. Like conditions of the *right ventricle* may dislocate apex slightly toward left side. The student will readily appreciate how a dilated right ventricle may dislocate the apex beat and prevent it from reaching the chest surface by the following experiment: Put one finger on the apex beat and another on the pulse of some artery; now suspend respiration, and, as the sense of suffocation approaches, the apex grows indistinct and may even disappear while the pulse is not manifestly affected, showing that the left ventricle is still contracting efficiently. When fluid and air are present in the pleural sac the dislocation of the apex is greater in accumulations on the right than on the left side. In dislocation from the causes last mentioned the heart is pushed "*in toto*" to one side.

The normal breadth of apex beat may be covered by the ungual phalanx of index finger and measures about 1 inch.

Increase in breadth occurs when heart is approximated to chest wall; *e. g.* contraction of lung, deformities of thorax and cardiac dilatation.

The normal force of apex beat is determined by palpation in health. *Increase in force* is observed in increased cardiac activity with or without (fever, exercise, etc.,) organic change.

When heart is dislocated toward chest-wall, there is an apparent increase in force. *Permanent increase in force and breadth* is the most important sign of hypertrophy of the left ventricle.

Diminished force of apex beat may exist in health, owing

to narrow intercostal spaces, increase in the coverings of the chest-wall, and from other causes not always understood.

Diminished force in disease is observed in emphysema, fluid in pleura or pericardium (which act by dislocating heart from chest-wall), and degeneration of heart-muscle.

Disappearance of apex beat is an important sign of beginning exudation in pericarditis. In this affection, the weakness of the apex beat is in marked contrast with the comparatively strong radial pulse, and the latter, in exudative pericarditis, is a good index of cardiac activity. *Fluid in pericardium* will usually dislocate apex downwards, for the following reasons: 1. The weight of heart is greater than the fluid, the latter occupying the upper, the heart, the lower part of pericardial sac. 2. Diaphragm and heart are pushed downward owing to the weight of the fluid.

The time of apex beat is usually synchronous with the pulse in the carotid and radial arteries.

The synchronism of apex beat and carotid pulse may be noted by placing one finger over the carotid artery, the other over the apex beat at the same time. Apex beat corresponds with contraction of the heart, *i. e. systole.* When the heart is irregular in action, and *murmurs* are heard, it is often difficult to say whether the murmurs are systolic or diastolic; by remembering that the carotid pulse is synchronous with the systole of the heart, the time of the murmur may be recognized. The *Carotid pulse* is not in reality coincident with the apex beat, inasmuch as the blood requires a certain time (0.093 of a second) to reach the artery.

Duplication of the apex beat is present when two apex beats can be felt corresponding with one carotid pulsation. Present in *mitral lesions*, and it is supposed that *hemisystole* exists, *i. e.* both ventricles do not contract at the same time.

The *cardiograph* is an instrument for registering the apex beat.

Abnormal Pulsations.—When present at the base of the heart in the second intercostal space to the right or left border of the sternum they originate respectively from the *aorta* or *pulmonary artery.* They are more often felt than seen. Systolic pulsations may indicate *aneurism* of these vessels.

Patella has recently directed attention to visible pulsations occurring over the pulmonary artery in *anæmia.*

Pulsations about the heart region occur in empyema of the left side (*empyema pulsans*) and in aortic aneurisms.

A *diastolic impulse* may be felt over the aorta or pulmonary artery, more frequently over the latter, and is due to the closure of the semilunar valves. When the heart and lungs are normal, it is not felt. It is usually observed when the right ventricle is hypertrophied or when the lung covering of heart is diminished or the lung consolidated.

Systolic retractions in cardiac region, when observed in two or more intercostal spaces, may indicate *adhesive pericarditis* with *mediastino-pericarditis*.

Thrills in the heart region are sensations felt by the hand similar to that perceived upon stroking the back of a purring cat. They are also called *frémissement cataire* or *purring tremors*.

Time and significance of thrills: 1, *Presystolic*; 2, *Systolic*; 3, *Diastolic*. They correspond with *murmurs* and are heard loudest in the same situations. They are nearly always indicative of a *valvular lesion*.

Pericardial thrill occurs in *pericarditis* and is caused by the rubbing together of the roughened layers of the pericardium.

TABLE OF THRILLS.

I. *At apex beat.*	1. *Systolic thrill*—Mitral insufficiency. 2. *Diastolic or presystolic thrill*—Mitral stenosis.
II. At the 2d right intercostal space, close to border of sternum.	3. *Systolic thrill*—Aortic stenosis. 4. *Diastolic thrill*—Aortic insufficiency.
III. At the right sternal border at 5th and 6th costal cartilage.	5. *Systolic thrill* — Tricuspid insufficiency. 6. *Diastolic or presystolic thrill*—Tricuspid stenosis.
IV. At the 2d left intercostal space, close to border of sternum.	7. *Systolic thrill*—Pulmonary stenosis. 8. *Diastolic thrill*— Pulmonary insufficiency.

Epigastric systolic pulsations are observed in the epigastric region when the diaphragm is low and the heart, particularly the right ventricle is approximated to the abdominal wall, or when

hypertrophy of the right ventricle is present. Also present in *abdominal aneurism.* Pulsations in this region may also be *transmitted* by the liver, enlarged stomach or tumors overlying aorta. When the abdominal walls are thin, aortic pulsations may be observed as a normal condition. The knee-elbow position will, as a rule, remove a transmitted pulsation, but if aneurism is present it will continue to pulsate distinctly.

PERCUSSION OF THE HEART.

Over the anterior surface of the heart uncovered by lung, *flatness* is obtained on percussion. This is in accordance with the general rule that all airless tissues, whatever their histological structure may be, give a pronounced dull sound on percussion. The anterior surface of the heart at its upper part and right half, being covered by lung tissue, yields, when the latter is not too dense, a dull sound on percussion. It will be seen from this, that two different forms of cardiac dullness are obtained; and furthermore, that where the lung tissue covering the heart is too dense, simple percussion will yield no results.

Forms of cardiac dullness: 1. Superficial or absolute. 2. Deep or relative. 3. Resistance of the heart.

Superficial or absolute cardiac dullness (Fig. 6), is obtained by weak percussion only. It represents that portion of the heart uncovered by lung tissue, and is the easiest to obtain.

A small portion of uncovered heart behind the sternum gives resonance on percussion, because the percussion blow is conveyed by the sternum to the adjacent lung structure.

The *superficial cardiac dullness* begins *above*, at the lower border of the 4th rib; the *right boundary* is at the left border of the sternum, the *left boundary* is formed by a line drawn from the 4th costal cartilage, curving convexly around, and ending at the apex beat. The lower boundary can not as a rule be determined by percussion, owing to the almost immediate contact of the left lobe of the liver with the heart. We may theoretically construct the lower border by drawing a line from the apex beat to the sternal insertion of the 6th rib on the left side.

The absolute cardiac dullness in children is relatively greater (large heart) than in adults. It may begin above in the 3d intercostal space, with its left border at or near the mammary line and the apex beat at the 4th intercostal space. In aged persons the area of cardiac dullness is diminished (emphysema.)

Area of cardiac dullness in respiration. Quiet respiration does not appreciably affect dullness. Forced inspiration diminishes, and expiration increases the area of dullness. Deep inspiration in certain persons may cause an entire disappearance of cardiac dullness.

Deep or relative cardiac dullness does not actually reproduce the entire size of the heart. Strong percussion is necessary. Like the absolute, it forms a triangular-shaped figure of dullness. The *upper border* is at the sternal insertion of the 3d left costal cartilage; the *right boundary* is at the right border of the sternum; the *left boundary* is formed by a line drawn from the upper border to the apex beat, exceeding the left boundary of absolute dullness by about one inch.

Resistance of the heart. This method, introduced by Ebstein, and determined by palpable percussion, is supposed to reproduce the actual size of the heart, a view which is justly questioned by competent observers.

Regarding the diagnostic value of the different forms of cardiac dullness, no unanimity of opinion exists. While percussion for the absolute dullness is generally employed owing to its comparitive simplicity, it is influenced by the position of the lung borders which, when consolidated or retracted, would give an apparent increase and when emphysematous an apparent decrease in the area of dullness. The determination of the relative cardiac dullness is largely influenced by the prejudiced wish of the observer. It does not reproduce the entire size of the heart, because the lung tissue covering the latter at certain parts is too dense, thus preventing the blow reaching the dullness beneath. It is decidedly inferior in accuracy to the former method, which, while presenting many errors, their careful elimination is possible by other physical signs.

The area of cardiac dullness is increased in hypertrophy and dilatation of the heart. The dullness is increased from above, downwards and outwards when the left ventricle alone is involved, while in hypertrophy and dilatation of the right ventricle the heart's dullness is broader and increased toward the right side. When both ventricles are implicated the dullness is increased transversely and longitudinally.

Fluid in the pericardial sac (*Hydropericardium*). The characteristic feature of this dullness is, that the outline of præcordial flatness is a *blunt cone.* An important sign is the change of the area of flatness in different posi-

tions. The dullness is first increased upwards on a level with the first intercostal space and laterally at the base. The dullness is usually extended to the left of the apex beat.

The *cardiac dullness* may be apparently increased even when the heart is normal as in *retraction of the lung.* In such a case, the lung borders about the heart undergo no change of position during respiration. An apparent increase of cardiac dullness may also occur in pleuritic effusions on the left side, or when the lung adjacent to the heart is infiltrated, or when tumors are present. Percussion in such cases is of no value in defining the cardiac dullness and recourse must be had to other signs.

The area of cardiac dullness is diminished in atrophy of the heart, emphysema of the lung and mediastinum, and pneumo-pericardium.

In *emphysema* the increased volume of lung causes it to envelop the area of cardiac dullness and replaces it by a vesiculo-tympanitic resonance. In *pneumo pericardium,* there is hyper-resonance over the præcordia, sometimes of a distinctly metallic character, when the patient is on his back; when he sits up this disappears.

Changes in location of the heart. In transposition of the viscera (*situs viscerum transversus*) the heart dullness and apex beat are found in the corresponding place on the opposite side. The heart may be dislocated to the right or left by *pleuritic fluid, adhesions, abdominal accumulations, tumors, etc.*

WEIGHT OF THE HEART.

Average male	10—12 oz. (Gray).
" female	8—10 oz. "

SIZE.

Length	5 inches.
Width	3½ "
Thickness	2½ "

THICKNESS OF HEART WALLS.

Thickness of right auricle	1 line (1-12th of an inch.)
" " left "	1½ lines.
" " right ventricle	2½ to 3 lines.
" " left "	4 to 5 lines.

CAPACITY OF VENTRICLES.

Capacity of right ventricle	2 fl. oz.
" " left "	2 fl. oz.

SIZE OF THE VALVULAR OPENINGS.

Aortic orifice	1 in.
Mitral "	1.8 in.
Pulmonary orifice	1.2 in.
Tricuspid "	2 in.

AUSCULTATION OF THE HEART.

Indirect Auscultation with the small attachment of the stethoscope is alone indicated. Auscultate patient in a state of physical and psychical rest, and in various positions. Auscultate during quiet respiration, then during inspiration and finally in expiration. It is often necessary to stimulate cardiac activity before auscultation is attempted, this may be done by directing patient to make various movements.

Acoustic Phenomena in Auscultation of the Heart. 1. Heart sounds. 2. Endocardial murmurs. 3. Exocardial murmurs.

Heart Sounds.—Two sounds are heard over the heart, the first corresponding with ventricular contraction, the *Systolic tone*, the second, the *Diastolic tone.*

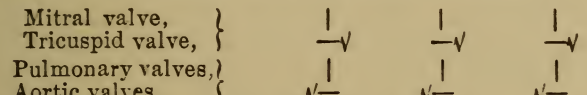
A very small interval of time is appreciable between the two sounds, with a distinct pause after the second sound. The first sound corresponding with the apex beat and carotid pulse is loudest at the apex, whereas the second sound is loudest at the base of the heart, and occupies the first portion of the period of diastole.

Difference Between the 1st and 2d Sounds. The first sound is longer, duller, and less clear than the second sound which is short, sharp and of high pitch.

The heart tones and murmurs are best heard at their points of origin. Auscultation can not always be practiced over the anatomical situations of the orifices of the heart owing to certain anatomical reasons. The *mitral valve* can not be auscultated at its anatomical situation owing to a covering of dense lung tissue, and the *aorta* is partially covered at its origin by the pulmonary artery. The following table gives the anatomical situations of the valves and points of auscultation. (See Fig. 6.)

NAME OF VALVE.	ANATOMICAL SITUATION.	POINT OF AUSCULTATION.
Mitral valve.	Upper border of the 3d left costal cartilage close to the sternum.	Apex beat.
Tricuspid valve.	Between the 3d left intercostal space and the 5th right costal cartilage.	Median line on a level with the 5th costal cartilage.
Pulmonary valves.	In the 2d left intercostal space close to the left border of sternum.	2d left intercostal space close to the left border of sternum.
Aortic valves.	Between the median line and 3d left costal cartilage.	2d right intercostal space close to the border of sternum.

Number and Origin of Heart Tones. Six sounds are heard over the heart. From each venous opening (*mitral and tricuspid valves*), a systolic tone, and from the arterial openings (*aorta and pulmonary*), a systolic and diastolic tone. Two sounds are heard at each opening. The second sound at the *mitral* and *tricuspid* is transmitted from the aorta and pulmonary artery. Over the ventricles the accent is on the first sound (*trochee*), over the aorta and pulmonary artery it is on the second sound (*iambus*). The second aortic tone is normally stronger than the second pulmonary tone. The following schema will show on what tone the accentuation falls:



Duration of the Two Sounds and Two Rests. The entire period of the heart action being represented by 10.

Duration of the first sound,	4
" " " " rest,	1
" " " second sound,	2
" " " " rest	3
	—10

Causes of the heart tones:

Apex beat (*mitral orifice*).

First tone: closure of the mitral valve and contraction of the ventricle.

Second tone: transmitted 2d aortic tone.

Lower end of sternum on a level with 5th costal cartilage (*tricuspid orifice*).

First Tone: closure of tricuspid valve and contraction of the ventricle.

Second tone: transmitted 2d pulmonary tone.

Second right (*aorta*) and left (*pulmonary artery*) intercostal space:

First tone: sudden tension of the aorta and pulmonary artery, and transmitted ventricle tone.

Second tone: closure of the aortic and pulmonary valves.

Observe the Following in Auscultation of the Heart Tones:

1. Rythm. 2. Strength. 3. Reduplication of the heart sounds.

Rythm. The rythm of the heart tones may be changed, and still be consistent with a normal heart, these changes being due to psychical causes, use of tobacco, sexual

excesses, or they may be congenital. Variations of rythm may, however, be due to degeneration of the myocardium, or associated with valvular disease.

Strength of the Heart Tones. Normally the intensity of the tones is influenced by the approximation of the heart to the chest-wall, and the density of the latter's coverings.

In children and emaciated persons, the tones are relatively loud. When the heart is pushed toward chest-wall, the tones are also loud. They are louder in the erect than in the recumbent position. During inspiration, the intensity of the tones is diminished, owing to lung covering heart, which is a poor conductor of sound; the contrary holds good when the lung is consolidated. The greater the activity of the heart's action, the louder the tones.

Strengthening of the First Heart Tone is observed when the work of the heart is increased, as in *hypertrophy;* also in *mitral stenosis.*

The cause of the strengthened first sound in *mitral stenosis* may be explained as follows: In consequence of stenosis, the left ventricle receives only slowly a small amount of blood; if, now, a normal systole of the ventricle occurs, a decided difference in the tension of the valve exists in systole and diastole, resulting in intensification of the systolic tone.

Strengthening of the 2d Aortic and Pulmonary Tones, if lasting, is a most important sign of *hypertrophy* of the left and right ventricles respectively.

Weakened First Sound occurs in degeneration of the heart musculature, emphysema and aortic insufficiency.

The *Systolic tone* at the apex is often *weakened* in *aortic insufficiency* and is owing to the great tension of the mitral valve at the end of diastole owing to the regurgitation of blood from the aorta, so that at the systole of the ventricle, the tension of this valve not being greatly increased the first tone is consequently weak.

Weakened Second Sound over the Aorta and Pulmonary Artery occurs in *stenosis* of these openings, and is due to the diminished blood pressure and vibration of the diseased valves. Also present in stenosis or insufficiency of the mitral orifice, and is due to the diminished tension of the semilunar valves caused by the small quantity of blood thrown into the aorta.

Reduplication of the Heart Tones is observed in unequal tension of the blood in both ventricles, which causes the valves on both sides to close at different times.

Reduplication of the tones may be physiological and dependent upon respiration. It may also be associated with pronounced disease of the circulatory apparatus.

Metallic Sounding Heart Tones are heard at times when lung cavities are in proximity to the heart; also in *pneumo-thorax, pneumo-pericardium, dilatation of the stomach, etc.*

ENDOCARDIAL MURMURS.

These murmurs have their origin within the heart, and are usually divided into *organic* and *inorganic murmurs*. Organic murmurs are usually produced by constriction of an orifice of the heart (*stenosis*) or an incomplete closure of the valves (*insufficiency*). The former is called an *obstructive*, the latter a *regurgitant murmur*.

Causes of Cardiac Murmurs.—When fluid passes through a tube which is suddenly narrowed, eddies are formed, which lead to audible murmurs. The more rapid the current the greater are the eddies and the louder the murmurs. As the cardiac orifices normally represent no decided narrowing, the blood produces no murmurs. If an obstruction (*stenosis*) to the outward flow of blood is present, then eddies are formed and murmurs heard. Murmurs due to obstructions are heard during systole. Murmurs arise in insufficiency of the valves in the following manner: After the blood has passed through an orifice, the valves close, but being insufficient, a portion regurgitates through the insufficient valves, causing a murmur.

The murmur of insufficiency is heard at the arterial opening during diastole, and during systole at the venous openings. Roughness of the arterial coats or of the orifices of the heart alone will not produce murmurs.

Cardiac murmurs are differentiated according to time, character, and position.

Time: Murmurs may occur during *systole* or *diastole*. A murmur peculiar to *mitral stenosis* and called a *presystolic murmur* is heard during the termination of diastole, or, occurring at the beginning of diastole, it is louder during the end.

Character: This has been variously described as blowing, rasping, sawing, etc. Murmurs of a musical character are sometimes heard and owe their origin as a rule to the vibration of a membranous substance, suspended in the blood stream.

Position: This refers to the point where murmurs are heard loudest. As a rule murmurs are most intense at

their point of origin. Murmurs are propagated in the direction of the blood current by which they are developed.

Axioms:—1. The character or intensity of a murmur is no index to the gravity of the lesion producing it. The loudest murmur may be produced by the smallest lesion and *vice versa*.

2. The intensity of a murmur is largely dependent on the activity of the heart. Faint murmurs may often be converted into loud murmurs after increasing cardiac activity by active exercise. Weakness of the heart may abolish a murmur previously distinct, the murmur reappearing after cardio-tonic medication.

3. A murmur is generally louder when the patient is lying down, then when he is standing. Always auscultate murmurs with the patient in various positions.

4. Murmurs are less loud in inspiration than expiration.

5. Strong pressure on the chest may cause the disappearance of murmurs, the pressure inhibiting cardiac action.

6. When the heart is rapid or irregular in action, it is difficult to determine the time of a murmur. Remember that systolic murmurs are synchronous with the carotid pulse. Also regulate the action of the heart with digitalis.

7. Systolic are usually louder though less prolonged then diastolic murmurs.

8. When murmurs are faint, the patient should suspend respiration during auscultation.

Coexistence of Several Murmurs.—This is frequent when insufficiency of the valves is combined with stenosis of the same orifice. In such a case murmurs would be heard during systole and diastole. When different orifices are involved at the same time it is difficult and even impossible to localize the lesions by auscultation alone.

Endocardial Inorganic Murmurs (*anæmic or hæmic murmurs*). The cause of these murmurs is yet *sub judice*. They have been attributed to disturbances in innervation, to *relative insufficiency of the valves*, *i. e.* the valves, without having undergone any anatomical change, are no longer able to close the enlarged orifices of the heart, and finally to diminution in the quantity of blood. In the latter condition the smaller arteries, veins and capillaries must accommodate themselves to the diminished quantity of blood, and contract; but the aorta and pulmonary artery cannot contract to the same degree; their diameter, in consequence, being proportionately larger than that of the orifices through which the blood enters them, murmurs are produced.

TABULAR VIEW OF CARDIAC VALVULAR MURMURS.

TIME OF MURMUR.	USUAL SEAT OF MURMUR.	OCCASIONAL SEAT OF MURMUR.	CAUSE OF MURMUR.
Aortic orifice —Systolic murmur.	2d right intercostal space close to the border of sternum.	Over the carotid arteries. The most widely diffused of all cardiac murmurs.	Obstruction to outward flow of blood through aortic orifice.
Mitral orifice —Systolic murmur.	Apex beat.	2d left intercostal space close to the border of sternum; may also be transmitted by chest-wall to the axillary border or beyond it.	Regurgitation of blood through mitral orifice into left auricle.
Pulmonary orifice—Systolic murmur.	2d left intercostal space close to the border of the sternum.		Obstruction to outward flow of blood through pulmonary orifice.
Tricuspid orifice—Systolic murmur.	Median line of sternum on a level with 5th costal cartilage.		Regurgitation of blood through tricuspid orifice into right auricle.
Aortic orifice —Diastolic murmur.	2d right intercostal space close to the border of the sternum.	In the middle of sternum. It may also be heard over the aorta and carotids.	Regurgitation of blood through aortic orifice into left ventricle.
Mitral orifice, —Diastolic murmur.	Apex beat.	Murmur rarely diffused. Usually limited to apex beat.	Obstruction to the flow of blood from left auricle to the left ventricle.
Pulmonary orifice—Diastolic murmur.	2d left intercostal space close to the border of the sternum.	Down right side of heart.	Regurgitation of blood through pulmonary orifice into right ventricle.
Tricuspid orifice—Diastolic murmur.	Median line of sternum on a level with 5th costal cartilage.	May be transmitted to the base of the heart or to the right axilla.	Obstruction to the flow of blood from right auricle into right ventricle.

Characteristics of anæmic murmurs:—

1. Usually soft and blowing in character, and not prolonged.
2. Occur during *systole* only, and usually systolic tone is heard.
3. Generally loudest at base of heart.

4. Accompanied with anæmic symptoms, and murmurs in the veins of the neck.

5. Unaccompanied, as a rule, by changes in size of the heart.

6. They frequently change their character.

7. Under appropriate treatment of the general condition, they disappear.

Anæmic murmurs are usually loudest over the *pulmonary artery*, a point where organic systolic murmurs are frequently heard. The chief means of differentiation between the two, lies in the fact that with organic murmurs we find *dilatation* and *hypertrophy of the heart*, which are usually absent in anæmic murmurs.

Exocardial or Pericardial murmurs. These murmurs are friction sounds produced by the rubbing of one surface of the pericardium upon the other when roughened by a fibrinous exudation.

Characteristics of pericardial murmurs:—

1. Unlike endocardial murmurs, which are limited to a certain phase of the heart's action, they may be systolic, diastolic, or both, or even presystolic.

2. They are increased in intensity upon pressure with the stethoscope, which maneuver facilitates the friction between the pericardial layers.

3. During deep inspiration the lung approximates the layers of the pericardium, thus increasing pericardial murmurs during this phase of respiration. Endocardial murmurs by the same act are diminished in intensity, because the interposed lung offers a poor medium of conduction to the chest-wall.

4. Pericardial murmurs are circumscribed, and, unlike endocardial murmurs, are not transmitted beyond the area of cardiac dullness.

5. Change of position exerts a greater influence on the character of pericardial than endocardial murmurs. Pericardial murmurs are rendered especially distinct when the patient is in the sitting posture, with the body inclined to the left side.

6. Pericardial murmurs give the impression of being of superficial origin.

7. Pericardial murmurs frequently change their character, thus being unlike endocardial murmurs, the character of which is almost constant.

8. Pericardial murmurs are rough, grating to and fro, or rubbing and scratching sounds.

Extra-Pericardial Murmurs.—These sounds are with difficulty distinguished from pericardial sounds proper. They occur when the pleura or peritoneum adjacent to the heart is roughened. They differ from pericardial murmurs by being dependent more on the movements of respiration than of the heart, and they may at times be made to disappear by suspending respiration.

Palpitation of the Heart.—This common complaint is often

mistaken by the patient for some organic heart affection. Among the causes are weakness of the heart muscle (fatty degeneration); excessive use of stimulants (tea, coffee, tobacco), and psychical disturbances. It is also frequent in so-called nervous patients. It is manifested in the main by paroxysms of increased frequency of the heart action, unattended by any increase in the heart dullness. When associated with systolic murmurs the latter are usually of anæmic origin. Palpitation as a functional disturbance is not associated with dyspnœa and venous stasis or other symptoms peculiar to organic affections of the heart.

Puncture of Pericardium.—This may be practiced as an aid to diagnosis or in the removal of large effusions. The point of election, according to Roberts, is in the 5th intercostal space from 2 to 2¼ inches to the left of the median line of the sternum. Aside from injuring the heart or puncturing the pleura, the selection of this point prevents injury to the internal mammary artery which runs from ¼ to ½ an inch from the left border of the sternum.

DIAGNOSIS OF CARDIAC DISEASES.

Hypertrophy of Left Ventricle.—*Symptoms*: Increased tension of the radial pulse, pulsation of temporal and carotid arteries, vertigo, headache, florid face, ringing in the ears, epistaxis and insomnia. *Physical signs. Inspection*: Bulging of præcordial region, apex very forcible and extended downwards and to the left. *Percussion*; increased dullness downwards and to the left. *Auscultation*; loud heart tones with accentuation of 2d aortic tone.

Hypertrophy of Right Ventricle.—*Symptoms*: Stasis in the pulmonic circulation leading to dyspnœa, hæmoptysis and bronchitis. *Physical signs. Inspection*; Apex beat diffused and marked in the epigastrium. *Percussion*; increased cardiac dullness to the right. *Auscultation*; accentuation of 2d pulmonary tone.

Dilatation of the Heart.—*Symptoms*: Peculiar to a feeble circulation, turgid veins, feeble pulse, dyspnœa, œdema and effusions in the internal cavities and symptoms indicative of congestion of the viscera. *Physical signs. Inspection*: Apex beat diffused and very feeble. *Percussion*; Cardiac dullness increased to the right or left according to side of heart involved. *Auscultation*; Cardiac tones are very feeble and indistinct.

Pericarditis; *1st or dry stage.*—Apex beat tumultuous and irregular; friction fremitus felt and friction sound heard.

2d or stage of effusion.—Apex beat feeble and fluttering; cardiac dullness of a triangular form. Heart sounds, feeble or absent over the area of dullness, but are heard above the line of effusion distinctly and loud.

3d or stage of absorption.—Disappearance of dullness, and reappearance of heart tones and friction sound. If effusion is not absorbed, the physical signs of 2d stage continue.

Aortic Regurgitation.—*Symptoms*; pulsation of the arteries, *pulse* is forcible, but recedes from the finger (*waterhammer, or the Corrigan pulse*); capillary pulse. Sphygmographic tracing characteristic. (*See pulse*).

Physical signs.—Bulging præcordial region, apex beat extended to the left and downwards, increased cardiac dullness to the left. Diastolic murmur over the aorta. Systolic murmur at times at apex, and accentuation of 2d pulmonary tone.

Aortic Stenosis.—Vertigo frequent. Apex beat extended to the left and downwards. Cardiac dullness increased to the left. Loud systolic murmur over aorta; also heard over other ostia, although feeble; 2d aortic tone absent.

Mitral Regurgitation.—Pulse irregular. Apex beat dislocated downwards and outwards and diffused. Apex beat is feeble, and a systolic thrill is felt. Increased dullness peculiar to hypertrophy and dilatation of right ventricle. Systolic murmur at apex and over pulmonary artery, traceable to axillary region and angle of scapula.

Mitral Stenosis.—Apex beat felt in epigastrium and to the right of sternum. Presystolic thrill at apex. Percussion same as mitral regurgitation. Prolonged presystolic murmur at apex, and usually localized.

Tricuspid Regurgitation.—Increased cardiac dullness to the right, systolic murmur over tricuspid valve, weakened 2d pulmonary tone, venous pulsation. (*See examination of the veins.*)

Tricuspid Stenosis.—This is the rarest of all heart diseases. Venous stasis occurs, and the symptoms are the same as tricuspid regurgitation.

Diseases of the *pulmonary valves* are usually congenital, and characterized by pronounced cyanosis, dilatation of the right side of heart, and murmurs, according to character of lesion, best heard over pulmonary artery.

CHAPTER VII.

EXAMINATION OF THE ARTERIES AND VEINS.

The Arteries.—*Methods*: *Inspection, palpation, percussion, auscultation.*

Inspection. Pulsation of the larger arteries is not usually discerned in healthy persons. Pulsations of the arteries of the neck are seen when the heart is very much increased in action. In *hypertrophy of the left ventricle*, these pulsations are permanent and pronounced.

Capillary pulse. In healthy individuals, the capillary blood-vessels at the roots of the nails often show systolic redness and diastolic paleness. This so-called "capillary pulse" becomes exaggerated in diseased conditions, notably in *aortic regurgitation.* The explanation of this phenomenon is as follows: In consequence of the hypertrophy of the left ventricle of the heart, an increased quantity of blood received by regurgitation from the aorta and left ventricle, is thrown into the relatively empty aortic system; then follows a rapid emptying of this system toward the capillaries and the heart.

Ophthalmological examination often shows pulsation of the retinal blood-vessels. At times, pulsations of the *liver* and *spleen* can be felt. F. Mueller has lately called attention to pulsations of the *soft palate*, which he claims are similar in origin and significance to the capillary pulse. Capillary pulsation may be demonstrated by making lines with some blunt pointed instrument on the skin of the forehead, and the lines are seen to become alternately pale and red.

Palpation. In many of the arteries, notably the radial and temporal, the walls are thickened and rigid, and cartilaginous-like plates are felt. This condition is peculiar to *arterio sclerosis*, and is due to *calcification* of the middle arterial coat.

Auscultation. This is accomplished by means of a stethoscope, avoiding pressure of the artery.

Normal auscultation. Over the *carotid* and *subclavian* arteries, two tones are heard. The first is the transmitted *1st aortic tone*, the second, the transmitted *2d aortic tone*.

Over the *abdominal aorta* and *femoral arteries*, one tone (*due to tension of the artery*) is at times heard, although as a rule no sounds are normally heard. *Murmurs* heard over arteries are usually pathological. The student will do well to familiarize himself with certain errors consequent upon unskilled auscultation of the arteries. For study, select the *brachial artery* at the elbow joint; now make moderate pressure with the stethoscope on the artery, which will develop a *murmur* (*pressure murmur*). If the pressure is increased to a certain point, a short, sharp *tone* is produced (*pressure tone*). Auscultation of the *carotid artery* is practiced at almost any part of its accessible course and of the *subclavian artery* above the clavicle, or below in the *fossa of Mohrenheim*.

Arterial Murmurs. In *aortic stenosis* a murmur replaces the first tone over the carotids. The second tone over the carotids and subclavian is replaced by a murmur in *aortic regurgitation*. Murmurs originating at the mitral and tricuspid valves can likewise be transmitted to the arteries. Murmurs from the aorta are best transmitted by the right, and murmurs from the pulmonary artery by the left carotid.

Locally Produced Arterial Murmurs occur whenever an artery in its course is dilated or narrowed. The most important of these murmurs is the *aneurysmal murmur*, which may be systolic, diastolic, or both.

Differences in Symmetrical Arteries. In health symmetrical arteries show the same pulse qualities, unless anatomical anomalies exist. Any mechanical obstruction, whether due to emboli, aneurisms or tumors compressing an artery will make a comparative change in the qualities of the pulse.

Examples: If an aneurism involving the arch of the aorta is situated between the *innominate* and *left subclavian artery*, then the pulse of the right radial and carotid arteries can be felt sooner than in the same arteries on the left side. Again, if the aneurism lies between the *left carotid* and *left subclavian arteries*, the pulse can be felt earlier in both carotids and in the right radial artery than in the left radial and femoral arteries.

Paradoxical Pulse. This occurs when the radial pulse disappears either during inspiration or expiration. It is caused by adhesions between the subclavian artery and the pulmonary pleura.

When the shoulders are pressed backwards and downwards with the hands on the gluteal regions, and deep inspiration is made, the *radial pulse* on both sides can be made to disappear. This is explained by compression of the subclavian arteries by the first rib. When the first rib has become rigid in consequence of an *ossifying perichondritis*, a condition peculiar to *phthisis*, then notwithstanding deep inspiration, the radial pulse is still present.

Percussion is of limited application, although it is useful in defining the situation of an aneurism.

Subclavian murmurs are heard over the subclavian arteries either during inspiration (more often) or expiration.

These murmurs are frequently present in *phthisical persons*, and the explanation of their occurrence is similar to that of the paradoxical pulse, viz. adhesions between the subclavian artery and pulmonary pleura. Fuller found the subclavian murmur present 12 times in 100 healthy persons.

Brain murmurs are heard in children from the 3d month to the 6th year by auscultation of the head. Over the regions of the fontanelles the murmurs are loudest. They are physiological and are transmitted from the carotids.

Pathological arterial tones are heard over arteries where tones are normally absent. In *aortic regurgitation*, in consequence of sudden tension of the arteries, tones are heard corresponding with the pulse in peripheral arteries.

Thyroid arterial murmurs are present in all enlargements of this gland.

THORACIC ANEURISM.*

Symptoms. Pain is nearly always present. Pressure on various nerves, on the trachea, œsophagus, etc., will produce characteristic symptoms.

Physical Signs.—*Inspection.* Until the aneurism has attained a certain size nothing is observed. Later, *bulging* is apparent upon the surface of the chest involving the upper part of, or immediately to the right, of the sternum. If the *transverse portion* of the aorta is involved then *pulsation* is observed in the *supra sternal fossa.* The pulsation of an aneurism corresponds with the systole of the heart and is *equally distended in all parts.*

* Schnell has recently directed attention to an apparatus called the *Aneurysmatoscope*, for the diagnosis of aneurisms of the *descending aorta.* It consists of an ordinary soft rubber stomach tube closed at its lower end, and containing at its upper end a glass tube. The whole is then filled with a colored fluid up to the latter. When the tube is introduced into the œsophagus, the pulsations of the aneurism are transmitted to the fluid in the tube, and can thus be recognized.

Palpation. Careful palpation will discover an *abnormal pulsation* usually systolic in time, even before the aneurism reaches the surface of the chest. A *systolic or diastolic thrill* may accompany the pulsation.

Percussion yields a *dullness* even in the early stages of the aneurism, which may be limited to the *manubrium* or to the right of the sternum. The dullness may be continuous with or distinct from the cardiac dullness.

Auscultation. One or both heart sounds may be heard; or a *systolic or diastolic murmur* or both. At times no sound or murmur is heard.

THE VEINS.

Inspection and palpation. 1. *Increased distension of the veins.* 2. *Abnormal movements of the cervical veins.* 3. *Thrombosis.*

Increased Distension. This is present when the return of the venous blood to the heart is rendered difficult in consequence of *general* and *local causes.*

General causes: Diseases of the heart and lungs.

Whenever the power of the right auricle and particularly of the right ventricle is reduced, these cavities are incompletely emptied and the return of venous blood to the heart by means of the superior and inferior venæ cavæ is prevented. The return of venous blood to the heart is facilitated by the *aspiration of the thorax* and this is dependent on the elasticity of the lungs. Any affection diminishing lung elasticity conduces to venous stasis and abnormal distension of the veins.

Local causes: Thrombosis of or compression of the veins. *Abnormal movements of the cervical veins* may be *respiratory* or *pulsatory.*

During inspiration the return of venous blood to the heart is facilitated, whereas during expiration a physiological hindrance exists. When the veins are filled with a normal quantity of blood, no respiratory movement is observed. It is only when abnormal distension is present, that this movement is discerned. The veins may swell only in *expiration*, in *mediastino-pericarditis* owing to a diminished caliber of the veins in inspiration.

Pulsatory movements are dependent on cardiac activity and show the same rythm as the latter. The pulsatory movement may be *communicated* or *develop* in the vein itself.

The first is communicated to the internal jugular vein by the pulsation of the carotid artery especially when the latter pulsates violently.

If the pulsation of the vein is communicated, pressure with the finger on the vein in the middle of the neck, will cause the pulsations to cease in the veins below the finger. If the carotid artery is effectually compressed the pulsation in the vein will also disappear showing that it is communicated from the artery.

True venous pulsation is observed in the jugular veins independent of any impulse communicated by the carotid artery, and is caused by regurgitation of blood during the systole of the heart into the superior vena cava and jugular veins. This pulsation is pathognomonic of *tricuspid insufficiency.*

Unlike the communicated pulsation, pressure with the finger on the vein, only increases the pulsation below the finger. The true venous pulse is most pronounced in the *right internal jugular vein.*

Venous Pulse of the Liver. This is a pulsation of the liver present in *tricuspid insufficiency* and is caused by regurgitation of blood into the inferior vena cava.

In *aortic regurgitation* pulsation of the liver has been observed and is caused by an abnormal distension of the hepatic arteries.

Auscultation.—Venous tones occur when blood regurgitates with a certain force from the heart into the veins, thus bringing the valves of the veins into sudden tension and causing a tone. Present in tricuspid insufficiency.

Venous murmurs when present are heard most frequently over the internal jugular vein of the right side at its junction with the subclavian to form the innominate. These murmurs are usually continuous *(continuous hum or bruit de diable)*, and are well marked in *anæmic* and *chlorotic* persons.

Pressure on the vein with the stethoscope must be avoided.

The venous murmur is not confined to either the systole or diastole of the heart, but persists through both. It is loudest during inspiration and is increased in intensity when the head is turned to the other side.

CHAPTER VIII.

THE PULSE.

The *pulse* is the expansion of an artery produced by a wave of blood set in motion by the passage of blood into the aorta at the systole of the ventricle. Palpation determines: 1. The frequency. 2. The rythm. 3. The quality of the pulse.

Frequency of the Pulse. In the healthy adult there is an average of 70 beats in a minute, in children 100–140, and in old men, 70–90, or more.

The frequency of pulse is greater in the female than in the male. The *daily variation* in the frequency of the pulse corresponds with a like variation in the body temperature. It is *most frequent* between noon and evening, and is *least frequent* in the early hours of the morning. The influence of *position* is such that it is most frequent standing, and least frequent in the recumbent posture. External *temperature* when high increases, when low diminishes the frequency. Increased muscular exertion, acceleration of respiration, and psychical activity also increase the frequency of the pulse.

Pathological Pulse Frequency. A slower movement of the pulse (*pulsus rarus*), and acceleration of the pulse (*pulsus frequens*) are distinguished.

Pulsus Rarus (*bradycardia*). Observed in conditions leading to an irritation of the vagus, or paralysis of the intra-cardiac ganglia and of the sympathetic. Also, in increased cranial pressure (*meningitis*), icterus (from the action of the gall acids on the cardiac ganglia), stenosis of the aortic and mitral orifices, degeneration of the myocardium (fatty heart); in colic, and after administration of digitalis.

Pulsus Frequens (*tachycardia*). Observed in paralysis of the vagus, irritation of the sympathetic and affections of the cardiac ganglia; in excessively increased cerebral

pressure (*last stage of basilar meningitis*), acute diseases of the heart (peri and endocarditis), and in valvular heart diseases when compensation is disturbed. Excessive rapidity (over 160) is a sign of heart-weakness *(collapse.)*

Paroxysmal tachycardia is also a symptom of functional diseases of the heart. In *hysteria* and *Graves' disease* the pulse is also increased in frequency.

Rythm of the Pulse. Irregularity of the pulse (*pulsus irregularis*) when observed in children and adults, nearly always indicates disease of the heart or brain. In advanced age irregularity of the pulse is of no importance.

Forms of Pulsus Irregularis: Pulsus alternans, a low pulse-wave following a high one; *pulsus bigeminus*, a long pause after two beats; *pulsus trigeminus*, a long pause after three beats; *pulsus paradoxus*, the pulse is smaller with each inspiration, or disappears (observed in stenosis of the air-passages, mediastinitis, and pericardial adhesions). *Retardation*, or *unequal size* of the pulse between symmetrical arteries, or between arteries of the upper and lower half of the body, is observed in stenosis of the arteries and aneurism.

Quality of the Pulse. In determining the quality, we consider: 1. Expansion; 2. Force and tension; 3. Size of pulse.

The arteries distend rapidly (*pulsus celer*) in hypertrophy of the left ventricle and in increased heart action. *Pulsus celer* is characteristic of *aortic insufficiency, contracted kidney*, and *exophthalmic goitre.* The arteries distend slowly (*pulsus tardus*) in old age (*senile pulse*), *aortic* and *mitral stenosis*, and in *aneurisms.* In considering the force and tension of the pulse, we speak of a *hard* (*pulsus durus*) and a *soft* (*pulsus mollis*) pulse. The hardness of the pulse is dependent on the tension of the arterial wall; the greater the tension the harder the pulse. Such a pulse is difficult of compression. A *hard pulse* is observed in hypertrophy of the left ventricle, and in spasm of the arterial wall, as occurs in lead colic. A soft pulse is easily compressed, and is found in cardiac degeneration, fever, and anæmia. In *atheroma* of the arteries, where the walls are infiltrated with calcareous salts, the pulse is *apparently* hard. The artery in such a condition can be rolled under the finger.

The *size of the pulse* is dependent on the force of the heart, the amount of blood in the artery, and the tension of its wall. A large pulse (*pulsus magnus*) is observed in *aortic insufficiency*, and a small pulse (*pulsus parvus*) in *aortic stenosis.* Sometimes the pulse is so slight as to be thready (*pulsus filiformis.*)

The Pulse in Diseases of the Heart.—

1. ***Mitral Insufficiency;*** no decided departure from the normal.

2. *Mitral Stenosis;* pulse small and irregular, frequency increased.

3. *Aortic Insufficiency;* pulsus celer, frequency normal or increased, usually regular.

4. *Aortic Stenosis;* pulse small, retarded, normal or diminished frequency, and regular, as a rule.

5. *Myocarditis;* pulse small and soft, irregular, frequency normal, diminished or increased.

A comparatively strong pulse, with feeble apex beat and heart-tones, is of great value in the diagnosis of *exudative pericarditis.*

SPHYGMOGRAPHY.

This is a method of graphically recording the characters of the pulse by means of the sphygmograph.

Normal Pulse Tracing.—(Fig. 9) We notice first an *ascending line* which is straight, and then a *descending line* which shows certain elevations. The ascending and descending lines meet above at what is called the *summit* of the pulse curve. Where the ascending line begins and the descending line ends is called the *base of the curve.* The ascending corresponds with the filling of the artery; the descending line with its collapse. The more rapid the flow and the more quickly the artery distends the more vertical the line.

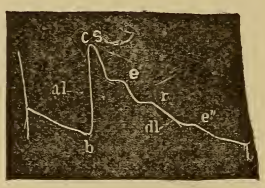


Fig. 9.

Fig. 9. Normal pulse. al. Line of ascent. dl. Line of descent. cs. Summit of curve. b. Base of curve. r. Elevation of recoil. e′ e″. Elastic elevations.

The descending line is broken in its descent by elevations which are distinguished as the *elevations of elasticity* and the *elevation of recoil.* The *elevation of recoil* is situated about half way down the descending line and is produced by the column of blood after impinging on the already closed aortic valves, producing a new wave.

The *elevations of elasticity* are smaller than the elevation of recoil (also called *dicrotic wave*) and are produced by vibration of the tense elastic arterial wall. In a normal pulse curve two elastic elevations are found, one above the other below the dicrotic wave.

Pathological Sphygmographic Tracings.—*Anacrotic pulse.* This is present when elevations are found on the ascending line, and indicate some abnormality in the arterial distribution of the blood.

Fig. 10.

It is found in any disease of the heart or arteries which allows of only spasmodic entrance of blood into the latter.

The greater the decrease in arterial tension the more pronounced is the *elevation of recoil.* When the tension of the artery is increased *(lead colic, acute and chronic nephritis)* the elastic elevations of the artery are pronounced, and the elevation of recoil is but feebly represented. The elevations of elasticity may disappear when the arterial tension is diminished.

Dicrotic pulse. This is characteristic of fever, which decreases arterial tension, thus bringing into prominence the recoil elevation, and causing the disappearance of the elastic elevations.

Many forms of dicrotic pulse are distinguished: 1. *Subdicrotic* (when the *elevation of recoil* appears before the descending line has reached the base of the curve). 2. *Dicrotic* (after it has reached the base of the curve). 3. *Hyperdicrotic* (when the recoil elevation belongs to the ascending part of the next wave). 4. *Monocrotic* (when no recoil elevation can be recognized.)

Pulsus tardus (senile pulse). A slow ascending line, with round and broad summit and no elevations on the descending line. Observed in *atheroma* of the arteries.

Pulse Curve in Valvular Lesions of the Heart.—*Aortic Stenosis.* The ascending line is more sloping owing to the retarded entrance of blood into the arteries, and the recoil elevation

is absent or imperfectly marked because the arteries are incompletely filled. *Aortic regurgitation*: the ascending line is high and vertical, the summit pointed and the dicrotic wave absent. *Mitral lesions*: in *mitral obstruction*, the pulse curve corresponds with diminished arterial tension, whereas in *insufficiency* the tracings are varied in relation to the amount of hypertrophy present.

Pulse Curve in the Veins.—This is the reverse of that in the arteries, the ascending line rising slowly, whereas the descending line falls quickly.

In *tricuspid insufficiency* the *venous pulse* begins in the diastole of the heart and reaches its maximum in the systole, whereas the venous pulse occurring with a normal tricuspid valve falls immediately before the beginning of the systole. The venous pulse in tricuspid insufficiency results from the blood wave, which is thrown back during the systole of the heart through the insufficient orifice into the auricle and thence into the venous system.

The clinical value of the sphygmograph cannot be denied. It not only confirms the evidence obtained by digital examination of the pulse, but makes evident certain qualities not appreciated by the fingers. It furnishes a record for future reference, and is almost indispensable for class instruction. Various modifications of Marey's sphygmograph have been introduced, one of the latest and best, being that of Ludwig.

CHAPTER IX.

THE BLOOD.

The whole *quantity* of blood in an adult is about $\frac{1}{13}$ of the body weight (10 lbs), whereas in new-born infants it is $\frac{1}{19}$ of the body weight. *Specific gravity* varies between 1045 and 1075. *Reaction* is alkaline.

Color of the Blood.—The blood removed from a healthy person is lighter when it contains much oxygen (*arterial*) and dark when oxygen is deficient (*venous*).

The blood of dyspnœic persons is very dark. *Carbonic oxide poisoning*, cherry-red color; *anæmia* and *chlorosis* (hydræmia), watery; *leucæmia*, pale.

Hæmoglobin. Amount contained in 100 ccm. of blood is for man, 13 to 15 grams, in woman slightly less. On heating, hæmoglobin is resolved into albumen and hæmatin.

Teichmann's test for blood. Dried blood heated on an object-glass with one or two drops of *glacial acetic acid* to the boiling point to which is added a grain of common salt and then slowly evaporated will show the formation of brownish-yellow rhombic crystals (*muriate of hæmatin*) the so-called *hæmin*.

Quantitative estimation of hæmoglobin can be approximately and very satisfactorily determined by means of *Fleischl's Hæmometer*, which consists essentially of comparing the color of the blood dissolved in water with a wedge of colored glass marked in percentages by an empirically determined scale. The *hæmoglobinometer of Gowers*, consists of two slender glass tubes, one of which contains a preparation of carmine and glycerine jelly, colored to represent a dilution of 1 part of healthy blood in 100 parts of water, the other tube being graduated into 100 divisions, each of which is equivalent to the volume of blood taken, so that 100 divisions=100 times the volume of blood.

The absolute estimation of hæmoglobin can only be determined by quantitative spectral-analysis. *Hæmoglobin is diminished* in chlorosis, secondary anæmia and advanced cases of leucæmia.

Spectroscopical examination. Examination of the blood by means of the spectroscope is of diagnostic importance in determining the following: *hæmoglobinæmia, carbonic oxide and chlorate of potash poisoning.*

Hæmoglobinæmia (when the hæmoglobin escapes from the corpuscles and is suspended in the blood plasma). Blood removed by means of the cupping-glass is allowed to stand in a covered vessel for 24 hours. In this time the serum is separated from the clot and is yellow in normal blood and ruby-red in hæmoglobinæmia. The serum obtained after this method shows in the spectroscope two bands in the yellow and green between the d and e lines of Frauenhofer. If a few drops of *ammon. sulphide* are added to the solution, the oxyhæmoglobin becomes *reduced hæmoglobin* and only 1 band is seen between D. and E. (This is likewise the spectroscopical reaction of normal blood.)

Carbonic oxide poisoning. Two absorption bands between D and E but closer together than in normal blood. The addition of *ammon. sulphide* does not cause their disappearance (*because carbonic oxide hæmoglobin cannot be reduced.*)

Chlorate of potash intoxication. The chocolate-colored blood shows, besides the oxy-hæmoglobin bands, an absorption band in the red of the spectrum. The addition of *ammon. sulphide* causes the disappearance of the 3 bands and the appearance of 1 band peculiar to reduced hæmoglobin.

MICROSCOPIC EXAMINATION OF THE BLOOD.

Method. After providing a clean object and cover glass, cleanse the finger thoroughly and, with a needle or a lancet, make a wound sufficiently large to admit of the exit of a drop of blood without pressure; place the object glass on the blood drop and adapt the cover glass to it without pressure. We find normally in the blood, red and white corpuscles and blood plaques (*hæmatoblasts*). In disease the corpuscles may be altered in number and size.

Oligocythæmia (diminution in the number of red blood corpuscles). Observed in all forms of *anæmia.* In chlorosis, no diminution in the number of the red corpuscles is observed. Normally, there is in man *5 millions*, in woman 4½ *millions* of red corpuscles to a cubic millimetre.

Of all apparatus to determine the number of red blood corpuscles that of *Thoma-Zeiss* is the simplest and best. It consists of a glass pipette about 10 cm. long, which contains in its upper third a reservoir inclosing a glass bead; to the upper end is attached a rubber tube, which, being placed between the lips, causes the fluid to ascend to any desired height by aspiration. The pipette is

graduated 0.1, 0.5, 1, and so on to 101. In order to count the corpuscles, suck up with the pipette the escaping blood made by a deep puncture in the finger tip, until it reaches the mark 1. The point of the instrument is now wiped and the diluting fluid (3 % solution of chloride of sodium) is sucked up to mark 101; after the whole is well shaken, a drop is placed on the *counting chamber* and covered by a cover glass. The counting chamber is fixed to the object glass and is $\frac{1}{10}$ mm. deep and its floor is divided into microscopical quadrates; $\frac{1}{4000}$ cubic millimetre being the capacity of a quadrate. After counting the number of red blood corpuscles contained in 16 quadrates we can easily estimate the number contained in a cubic millimetre. If the blood has been sucked up to the mark 101, the blood dilution is 1 : 100. The number of counted blood corpuscles is first multiplied by 4000 ($\frac{1}{4000}$ being the cubic capacity of a quadrate) then with 100 (the dilution of the blood); the product divided by the number of quadrates counted, equals the number of corpuscles in a cubic millimetre of blood. The white corpuscles may be similarly counted; the addition of methyl-violet to the diluting fluid causes them, by taking up the coloring matter, to become more prominent.

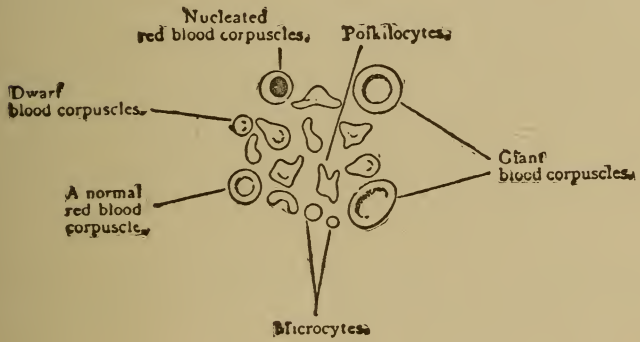


Fig. 11.

The Red Blood Corpuscle.—The size of the red blood corpuscles may be determined by comparison with the blood corpuscles from a healthy individual.

Macrocytes (Giant blood corpuscles). Found in *anæmia* and *progressive pernicious anæmia.*

Microcytes. Exceedingly small, bi-concave red blood corpuscles. Found in the blood of anæmic or hydræmic persons.

Poikilocytes. Red blood corpuscles of irregular form (pear, club or biscuit-shaped and other forms). Found in *severe anæmia.*

Nucleated Red Blood Corpuscles. Only recognized in stained preparations, and found in severe anæmia.

The White Blood Corpuscle.—The number of these corpuscles varies from 5,000 to 10,000 to the cubic millimetre and are temporarily increased after a hearty meal. The proportion between the white and red in health is 1 white to 335 or 600 red blood corpuscles; and more than 1 to 400 must be considered pathological. Not more than 5 white corpuscles normally appear in the field of vision when high objectives are employed. When more than 10 are seen the white corpuscles are increased in number.

Leucocytosis. A moderate increase in the number of leucocytes (1 to 100 red).

Leucæmia. An excessive increase in the number of leucocytes (1:50 or even 1:2).

Ehrlich divides the white blood corpuscles into: (1) *Lymphocytes*, they originate in the lymphatic glands and are about the size of a red corpuscle with a large round nucleus and little protoplasm. (2) *Monocular leucocytes*, larger than the red corpuscles with large oval nucleus and large protoplasmic body. They are the earlier stages of the development of the third. (3) *Polynuclear leucocytes*, they contain a divided nucleus and are deeply stained with aniline colors. (4) *Eosinophilous cells*, leucocytes containing in their protoplasm fatty granules which are colored an intense red on staining the dried blood with a 1% watery solution of eosin. The eosinophilous cells originate in the marrow of bones. In *myelogenetic leucæmia* they are very much increased being present in normal blood only in very small numbers. The *lymphocytes* are increased in leucæmia of lymphatic origin.

In all cases of leucocytosis the monocular and polynuclear cells are increased in number, but the eosinophilous cells are not. In leucæmia the eosinophilous cells are increased in number; so in doubtful cases of leucæmia the diagnosis can often be made by finding the eosinophilous cells.

Blood-plaques (*hæmatoblasts*).—These are colorless flat round discs about one-half the diameter of a red blood corpuscle. They are supposed to take an active part in coagulation. They may be demonstrated as follows: A drop of a solution of *methyl-violet* (1 to 5,000) is placed upon a punctured wound of the finger and mixed with the blood which is examined with high objectives.

Method of Staining Blood Specimens.—After puncturing the finger, place a drop of blood between two cover glasses and spread

it out in a thin film; next separate cover glasses and expose to the air until thoroughly dry. Then fix the hæmoglobin by heating the cover glasses for a few hours at a temperature of 212° F.; this may be accomplished by putting the glasses on a metal plate to one corner of which the heat may be applied. It is now ready for coloring and the following solution is usually employed.

Hæmatoxylin, 2 grams.
Alcohol,
Glycerine,
Distilled water, āā 100 grams.
Glacial acetic acid, 10 grams.
Alum in excess.

This solution is exposed to the light for 3 weeks and a few grains of *eosin* added. The cover glasses remain in the solution for 30 minutes and are then washed in water. The red blood corpuscles are stained red, the nuclei of the white and red corpuscles intensely black, the eosinophilous granules, red; whereas the protoplasm of the white blood corpuscles is only feebly stained. The stained preparation is examined with the oil immersion.

Melanæmia. Dark pigment scales either floating free in the blood or enclosed by amœboid movements of the white blood corpuscles. These scales may be deposited in the viscera. They are probably liberated by the disintegration of the red corpuscles and are found immediately after severe attacks of malaria.

Micro-organisms in the Blood.—1. *Bacillus anthracis.* May be present in the blood in *splenic fever* (malignant pustule, carbuncle) and is the sole cause of the affection.

The bacilli anthracis can usually be demonstrated without staining as immobile thick rods as long or twice as long as the diameter of a red blood corpuscle.

Spirochæte Obermeieri (spirillum). The spirilla are found in relapsing fever (*febris recurrens*) only during the febrile attacks in very large numbers and are the cause of the disease. They are long fine spiral fibres about 6 to 7 times the diameter of a red blood corpuscle in length, and are endowed with rapid movement. Their presence according to Gunther may be demonstrated as follows: The blood specimen prepared in the usual way on the cover glass is exposed for 10 seconds to a 10% solution of *acetic acid* in order to decolorize the red blood corpuscles, now remove the cover glass from the former

solution and blow the acid off with a pipette. Stain with a saturated solution of gentian violet in aniline water.

Bacillus Tuberculosis. May be found in the blood in cases of acute miliary tuberculosis. For detecting them, see *Sputum.*

Bacillus Typhosus. Often found in the blood in typhoid fever. It is a short ($\frac{1}{3}$ the diameter of a red blood corpuscle) thick rod with rounded ends.

Bacillus Mallei (glanders). Has been frequently found in this disease.

Plasmodium Malariæ. This organism is found in the blood in malaria inclosed in the red corpuscles as an amœboid mass frequently containing black pigment. The organism is best stained with methyl-blue. Whereas the etiology and biology of this organism is not definitely known, it has thus far only been discovered in the blood in malaria and its detection is of diagnostic importance in doubtful cases.

Animal Parasites (Hæmatozoa) in the Blood.—*Distoma Hæmatobium.* Occurs in the tropics and lives in the abdominal veins. It causes diarrhœa, hæmaturia and chyluria.

Filaria sanguinis hominis.

Chemical Changes in the Blood.—The alkalinity of the blood is diminished in severe anæmia, high fever, and diabetes. *Urea* may be increased in the blood in insufficiency of the kidneys. *Uric acid* is increased in *gout.* Normally *uric acid* is not demonstrable in the blood; but during and preceding the attack of gout it is greatly increased (0. 25—1.75%.) *Garrod's method* of detecting uric acid in the blood is as follows: To the fluid contents of a blister placed in a watch glass add 6–12 drops of diluted acetic acid (30%), then introduce into fluid a cotton fiber and let it remain for 24 hours, when, if uric acid is present, the crystals are deposited on the fiber and may be recognized by the microscope.

The blood after removal from the body is variously influenced in the rapidity of *coagulation* by different diseases. In health coagulation begins in about 9 minutes and is accelerated in chronic *disturbances of nutrition.* Fine fat granules are present in the blood (*Lipæmia*) in chyluria, diabetes and alcoholism. In *diabetes mellitus* the sugar (present in minimum quantities in normal blood) is increased.

Cholæmia. Biliary acids and pigment in the blood; the former dissolve the red corpuscles.

Uræmia. Accumulation of urinary constituents in the blood. The theory that urea is the essential toxic agent is no longer accepted.

DISEASES OF THE BLOOD.

Secondary Anæmia.—Observed in tuberculosis, severe dyspepsia, malaria, syphilis, carcinoma, anchylostomiasis (see *anchylostomum duodenale*), lead poisoning, nephritis, etc. The number of red blood corpuscles and the amount of hæmoglobin is diminished. The number of leucocytes is increased *(leucocytosis)*. Secondary, may pass into pernicious anæmia.

Progressive Pernicious Anæmia.—The number of red corpuscles is enormously reduced (a cubic millimetre may contain only 400,000) to a degree rarely attained even in the severest form of ordinary anæmia. Poikilocytes, macrocytes and microcytes are seen. The important diagnostic condition is the relative increase in the blood, of hæmoglobin. Prognosis is usually bad.

Chlorosis.—Observed usually in young girls. The essential condition is a great reduction in the amount of hæmoglobin, without reduction in the number of red, or an increase of white corpuscles. Poikilocytes may be present. Prognosis is usually good.

Leucocytosis.—This is a condition characterized by a temporary increase in the number of white blood corpuscles. Such an increase is physiological from 1 to 2 hours after the principal meal, when the relation to the red corpuscles may be 1:150 or even 1:100 (usual relation varies from 1:335 to 600). Leucocytosis is observed in many acute (pneumonia, erysipelas) and chronic diseases (carcinoma). In this affection the eosinophilous cells are *not* increased in number.

Leucæmia.—The white corpuscles are increased in number, the proportion of white to red exceeding 1:50 (the proportion may even reach 1:2). In the earlier stages of the disease it can only be diagnosed by the very *rapid increase* of leucocytes. Eosinophilous cells only occur in leucæmia. The number of red corpuscles and the amount of hæmoglobin is diminished. Crystals, like the asthma crystals (*see Sputum*) are sometimes found

in the blood. The following forms of leucæmia are differentiated: 1. *Lymphatic leucæmia; lymphocytes* increased and lymphatic glands enlarged. 2. *Myelogenic leucæmia;* eosinophilous cells, mononuclear cells, and nucleated red corpuscles are present and the bones (especially sternum and vertebræ) are painful on pressure.

Splenic (lienal) *leucæmia:* Blood appearance like the former and the spleen is enlarged. These forms are not always distinct and may be combined. The prognosis is usually bad.

Pseudo-leucæmia *(Hodgkin's disease).*—An affection characterized by cachexia, enlargement of the spleen and lymphatic glands. The leucocytes are not increased, although there is a slight decrease in the number of red corpuscles and in the amount of hæmoglobin.

CHAPTER X.

THE DIGESTIVE SYSTEM.

Odor from the mouth *(fœtor ex ore)* is observed in caries of the teeth, dyspepsia, etc. In *lead*, *phosphorus*, *alcohol* and *chloroform* intoxication the odor is often diagnostic. In *diabetes*, especially before or during diabetic coma the odor is likened to that of fresh apples.

The Lips.—The color acquaints us with the condition of the blood. Dryness and formation of crusts are observed in fever. In children *rhagades* at the angles of the mouth are characteristic of hereditary syphilis.

The Teeth.—In *diabetes mellitus* caries of the teeth may be pronounced. Bad teeth often lead to affections of the stomach. *Notched teeth*, *keratitis*, *fissure* at the lips, and *mucous tubercles* at the anus or mouth are characteristic symptoms of *congenital syphilis*. The teeth are *loose* in *scorbutus* and mercurial intoxication.

FORMULA OF THE MILK TEETH.—*(Finlayson.)*

$$\left.\begin{matrix} M_2\ C_1\ I_4\ C_1\ M_2 \\ \hline M_2\ C_1\ I_4\ C_1\ M_2 \end{matrix}\right\} \quad 20 \text{ in all}$$

TIME OF APPEARANCE.

Central incisors	7th month
Lateral incisors	9th "
First molars	15th "
Canines	18th "
Second molars	24th "

FORMULA OF THE PERMANENT TEETH.

$$\left.\begin{matrix} M_3\ B_2\ C_1\ I_4\ C_1\ B_2\ M_3 \\ \hline M_3\ B_2\ C_1\ I_4\ C_1\ B_2\ M_3 \end{matrix}\right\} \quad 32 \text{ in all}$$

TIME OF APPEARANCE.

Anterior molars	7th year
Central incisors	8th "
Lateral incisors	9th "
Anterior bicuspids	10th "
Posterior bicuspids	11th "
Canines	12th "
Second molars	12th to 14th "
Third molars	18th to 25th "

The Gums.—Spongy with submucous hæmorrhages in scurvy; swelling of the gums with salivation in mercurial poisoning. *Blue* or *blackish line* on the gums just above the teeth occurs in lead-poisoning. A *red line* on the gums is considered characteristic of a phthisical constitution.

The Tongue.—*Coated* in catarrhal affections of the stomach although in ulcer of the stomach and hyperacidity this is not usual. Soft white patches *(Muguet, Thrush)* due to the vegetation of the *oidium albicans* may be present; most often seen in children but likewise in adults suffering from exhausting diseases. The vegetable parasite may be seen with the microscope after removing the spots and adding liquor potassæ.

The tongue is enlarged in the different forms of *stomatitis*, inflammation (*Glossitis*), etc. Circumscribed swellings may be due to syphilis or carcinoma. Wounds and cicatrices on the tongue occur in epilepsy. In *hystero-epilepsy* wounding of the tongue rarely occurs.

Tremor in alcoholism and typhoid fever.

Strawberry tongue in scarlatina.

Saliva is an alkaline fluid (about 2½ pounds secreted in 24 hours), with a specific gravity of 1,002 to 1,006. It sometimes contains *sulphocyanide of potassium* (SCNK) which is recognized by adding a few drops of hydrochloric acid and a diluted solution of chloride of iron; a blood-red color appears, which is taken up on shaking with ether. Saliva may be further recognized by adding to it some diluted starch-paste and maintaining it at the body temperature for a few minutes when the conversion of starch into sugar can be shown by the usual test for the latter.

Saliva is increased (*Salivation, Ptyalism*) in all irritations of the mouth, after the use of mercury and in bulbar paralysis. *Diminished* in fever, diabetes, diarrhœa, etc. Reaction is acid in stomatitis and diabetes.

The Fauces.—Large *tonsils* with deep follicles indicate previous inflammations; prominent *white cicatrices*, syphilis. Chronic

ulcers on tonsils may be tuberculous or, more often, syphilitic in character. *Retro-pharyngeal abscesses* may form between the pharynx and the bodies of the vertebræ.

Anæsthesia of the throat is present in hysteria and alcoholism. Hyperæsthesia also present in drunkards.

EXAMINATION OF THE ŒSOPHAGUS.

Anatomy of the Œsophagus.—It commences at the lower border of the *cricoid cartilage* (opposite the 5th cervical vertebra), corresponding to the base of the xiphoid appendix, it terminates at the cardiac orifice of the stomach. It presents two or three slight curvatures in its course. At its commencement it is in front of the vertebral column, but inclines to the left of the column as far as the root of the neck, gradually passes in front of the column again, and finally, again deviates to the left as it passes forward to the œsophageal opening of the diaphragm. The average length of the œsophagus in adults is about 25 cm. (10 inches). Measurements of the œsophagus are usually reckoned from the upper incisor teeth. The distance from the upper incisors to the beginning of the œsophagus is about 15 cm. (6 inches). The distance from the incisor teeth to a point in the œsophagus opposite the *bifurcation of the trachea* is about 22 cm. (9 inches). The distance to the cardiac orifice of the stomach from the incisor teeth is in infants about 17 cm. (7 inches), and in adults about 40 cm. (16 inches). The narrowest parts of the œsophagus are at its commencement and at the point where it passes through the diaphragm.

The important neighboring structures of the œsophagus are : *trachea*, *bronchial glands*, *pleura*, *pericardium*, *aorta* (from the bifurcation of the trachea downwards), and the *inferior laryngeal nerve* (from the bifurcation of the trachea upwards.)

Methods of Examination : *Inspection*, *Palpation*, *Percussion*, *Auscultation.*

Inspection is usually limited to the neck portion of the œsophagus. *Tumors* and *diverticula* may be noted. For internal inspection of the œsophagus, a method known as *œsophagoscopy* has been introduced but with results negativing its universal practical application.

Palpation. — The finger introduced into the mouth cannot reach the œsophagus. Mediate palpation with sounds is the most important method of examination.

Introduction of Sound.—The sound must first be lubricated with glycerine (not oil). The patient sits on a chair with the head elevated; introduce the index and middle fingers of the left hand into the mouth of the patient down to the root of the tongue,

and with the sound grasped in the right hand, direct it to the ends of the fingers in the mouth, and with the ends of the fingers direct the tip of the sound downwards, and by elevating the right hand push it slowly in the same direction. *Physiological resistance* to the introduction of the sound is met with at the following points.

1. Wall of the pharynx. 2. At the commencement of œsophagus. Resistance is also encountered owing to the contraction of the œsophageal musculature (disappears after waiting a short time).

Dangers in the Introduction.—1. Perforation of the trachea (seldom). 2. Wounding or perforating the œsophagus. Abscesses and aneurism adjacent to the œsophagus may be penetrated by injudicious manipulation of the sound. *Never employ strong pressure with the sound when a resistance is encountered, and always use smaller sizes if the larger will not pass.*

In examinations with the sound *pain, the presence of diverticula, narrowing and dilatation* are noted.

Pain. May be produced by inflammation, ulceration or involvement of structures adjacent to the œsophagus.

Diverticula. When they are present the sound may at one sitting pass through the œsophagus and at another time it may meet with an obstruction.

Narrowing. The seat and degree of narrowing is determined. The seat of the narrowing *(stricture)* can readily be determined by measurements from the upper incisor teeth. The degree of narrowing is determined by the size of sounds necessary to pass the stricture. The nature of the narrowing may occasionally be determined by particles of tissue remaining attached to the sound after removal.

Dilatation is present above strictures and in paralysis of the musculature of œsophagus.

Percussion is only of very limited application. Diverticula in the cervical portion of the œsophagus when filled give a dull sound. If they are situated deeper, a dullness may be obtained along side the vertebral column. V. Ziemssen made dilatations of the œsophagus above the stricture evident by artificial distension with bicarbonate of soda and tartaric acid (which in combination yield carbonic acid gas) and obtaining in consequence a tympanitic or tympanitically dull sound over the diverticulum.

Auscultation is of little practical value.

A short murmur immediately after swallowing is heard over the entire course of the œsophagus during health. In stricture, this murmur may be delayed, weakened or absent. A longer murmur heard in the epigastric region about 7 seconds after swallowing (murmur of Kronecker and Meltzer) is of less importance.

EXAMINATION OF THE STOMACH.

Topography of the Abdomen.—If two circular lines are drawn round the body the one parallel with the cartilages of the 9th ribs, and the other with the highest points of the crests of the ilia, the abdominal cavity will be divided into three zones, *upper*, *middle* and *lower*. These zones are further divided by two parallel lines drawn from the cartilage of the 8th rib on either side down to the center of Poupart's ligament. Thus the three zones are subdivided into a middle and two lateral parts.

	MIDDLE REGION.	LATERAL REGIONS.
UPPER ZONE.	*Epigastric.*	*Right and left hypochondriac.*
MIDDLE ZONE.	*Umbilical.*	*Right and left lumbar.*
LOWER ZONE.	*Hypogastric.*	*Right and left inguinal.*

Anatomy of the Stomach. — Nearly the entire stomach lies to the left of the median line with the exception of the pylorus which is to the right of this line. The fundus lies under the left leaflet of the diaphragm extending as high up as the 4th intercostal space. The pylorus lies between the right sternal and parasternal lines, at the height of the tip of the ensiform cartilage. The cardiac orifice lies behind the sternal attachment of the seventh rib. The *lesser curvature* of the stomach and the pylorus are covered by the left lobe of the liver. The *greater curvature* in health rarely attains the umbilicus, being as a rule about 1 inch above the latter. That portion of the stomach which is uncovered by organs and directed to the surface of the chest and abdomen lies in the *half moon shaped space of Traube.*

INSPECTION AND PALPATION.

Normal condition. Inspection of the region of the stomach rarely shows any departure from the normal. After distension of the stomach by food a slight prominence in the epigastric region is noted.

PATHOLOGICAL CONDITIONS: *Pain, dilatation of the stomach, thickening of the stomach, increased peristaltic action of the stomach,* and *circumscribed tumors.*

Pain. On pressure it may be absent in all diseases of the stomach. It may be circumscribed or diffuse. In

acute and chronic catarrh of the stomach, pain if present is diffuse. In *ulcer of the stomach*, the pain is circumscribed. In order to render the stomach wall palpable it is often necessary to distend the stomach with carbonic acid gas. This is done, according to Frerichs, by administering a teaspoonful of bicarbonate of soda and of tartaric acid as separate doses, in water.

As this method of inflation often leads to dangerous results, it can be replaced with success by a method of recent introduction. This consists of introducing a stomach sound, through which air is forced in any desired quantity into the stomach.

If after the administration of bicarbonate of soda and tartaric acid no inflation of the stomach, but of the intestines occurs, then *incontinence of the pylorus* most likely exists. *Incontinentia pylori* may be functional or occasioned by destruction of the muscular tissue of the pylorus by *carcinoma* or *ulceration*.

Dilatation of the Stomach. When this is present inspection often shows a prominence in the upper part of the abdomen extending down to or below the umbilicus. Palpation may define the size of the stomach by its peculiar resistance. Of greater importance is the production of a *splashing noise* on palpation. Leube at one time determined the size of the stomach, by means of a sound, the point of which after introduction into the stomach was felt through the abdominal walls. He no longer practices this method, relying on *percussion* as a more certain means of diagnosis.

Thickening of the Stomach. This is determined by increased resistance on palpation ; present in hypertrophy of the musculature of stomach, a condition often accompanying dilatation. Circumscribed thickening should always awaken the suspicion of *carcinoma ; but contraction of the abdominal muscles must be excluded.*

Increased peristaltic action is determined by inspection and palpation. The movements of the stomach are usually directed from the cardiac orifice to the pylorus, at other times the peristaltic movements are irregular. They may occur spontaneously or must be induced by percussion or faradization of the abdomen. Increased peristaltic action is most frequently observed in *stenosis of the pylorus*, which leads to dilatation, and hypertrophy

of the musculature of the stomach. Kussmaul has shown that increased action may be purely a neurosis.

Circumscribed tumors are more often felt than seen. They usually indicate *carcinoma of the stomach*, and rarely, a *cicatrized ulcer*. Hypertrophy of the muscular coat of the stomach may lead to a circumscribed intumescence, but unlike carcinoma, which is hard with an uneven surface, it is smooth and less resistant.

Carcinomata of the stomach are seldom influenced by respiration unless adhesions exist between them and the liver, the respiratory movements of the latter being communicated to the tumor. Foreign bodies in the stomach and scybala in the colon may be confounded with tumors, but the differential diagnosis is usually easy.

PERCUSSION OF THE STOMACH.

The only portion of the stomach which can be percussed lies in the left hypochondriac and epigastric regions and comprises a part of the anterior wall. In order to define the stomach by percussion, the dullness of the liver is first determined to the right, the resonance of the lung to the left and above, and the tympanitic sound of the colon below. Between these organs the stomach gives a deep tympanitic sound which is with difficulty distinguished from the high tympanitic percussion note of the intestines. The size of the stomach is usually determined by percussing the lower border of the greater curvature, remembering that when it reaches below the umbilical line, the stomach is considered dilated. Percussion is best practised after inflation of the stomach, but as carbonic acid gas which is used for this purpose is dangerous we adopt the following method:

Introduce the stomach tube, remove the stomach contents and percuss abdomen while patient is standing; as a rule the boundary of the stomach cannot be made out; now introduce through the tube into the stomach about two pints of water and percuss again; a dullness is obtained, which disappears on removal of the fluid or when the patient lies down. In health, this dullness which represents the lower border of the stomach never reaches the umbilicus.

AUSCULTATION OF THE STOMACH.

Murmur of Deglutition. This is a murmur, normally heard in auscultating the epigastric region, occurring from 6–7 seconds after swallowing fluid. In insufficiency of the cardiac orifice (present in syphilis and phthisis) the murmur is heard immediately after swallowing. It is absent in obstruction at or above the cardiac orifice.

Splashing Noise. This is heard when fluid and gas are present in the stomach. It may denote a normal condition, but is frequently present in dilatation of the organ.

A sound likened to that of boiling water may be heard in fermentation of the contents of the stomach.

Gastroscope. This is an instrument used for the purpose of inspecting the interior of the stomach but it is not always adapted for practical use.

THE CHEMICAL ANALYSIS OF THE STOMACH AS A MEANS OF DIAGNOSING GASTRIC DISTURBANCES.*

Gentlemen: In the brief period of time at our disposal we can only, succinctly, review the late methods of diagnosis in diseases of the stomach. The artificial removal of the stomach contents is the only certain means of determining the character of the secretion and the degree of digestion. The removal of the gastric contents is best accomplished by the aid of the soft rubber tube, which I show you, working on the syphon principle. The introduction of the tube is simple. You ask the patient to make efforts at deglutition, and then gently and rapidly pass it into the stomach, which, when reached, is indicated by a black band on the tube. The real difficulty with which you will most frequently contend is the nausea and vomiting induced by the introduction of the tube. This sensitiveness of the fauces was formally combated by administering bromide of potassium for a few days before the introduction of the tube. This develops a moderate anæsthesia of the throat and diminishes the reflexes. A better method of preparation is that of painting the palate and the fauces with a solution of cocaine and lubricating the tube with a mixture of the same solution and olive oil. A difficulty in breathing is often complained of by the patient, which you can alleviate by asking him to take as deep breaths as possible.

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*Clinical lecture by ALBERT ABRAMS. Reported by ERNEST JOHANNSEN. Reprinted from *Occidental Medical Times*, April, 1889.

You will now ask the patient to cough violently or, if this does not suffice, to forcibly compress his abdominal muscles, when a sufficient amount of fluid for chemical analysis will usually be ejected from the tube. If then no fluid returns, connect your tube with a funnel, introduce some water into the latter, then hold the funnel over the patient's head until the water enters the stomach, but before all the fluid has left the funnel, lower it quickly and you will then get the contents of the stomach, plus water. Particles of food may stop up the orifice of the syphon. When this is suspected ask the patient to cough, or pour more fluid into the funnel, which will renew the suction action of the syphon.

After the ingestion of a meal, there is first a digestive period called the *amylolytic*, which lasts, on an average, three-quarters of an hour. After this time free hydrochloric acid is present, the amylolytic digestion ceases, and a peptonization of the albumen begins. The maximum degree of acidity (0.15 to 0.2 per cent. of hydrochloric acid) is attained about five hours after eating. During the entire period of digestion, the contents of the stomach, by peristaltic action, slowly pass into the duodenum. About six hours after eating, the stomach should be practically empty, and its contents should possess a neutral reaction. A diminished amount of gastric secretion interferes with the digestion of albumen, an excess (hyperacidity) interferes with amylolytic digestion, as the latter can only occur in the absence of free hydrochloric acid.

For the purpose of determining the duration of digestion I have given this patient a meal composed of soup, bread and beefsteak. Lavage of the stomach practiced seven hours after consuming such a meal would show, in normal digestion, that organ to be practically empty or containing a few fragments of food: under these conditions we would conclude that digestion is normal. We could further assume that there is probably no diminution of secretion, and absolutely, that the stomach has no difficulty in emptying itself. Should the stomach seven hours after the ingestion of a meal contain a considerable amount of food, then digestion is delayed or the viscus has some difficulty in emptying itself. This difficulty may occur in dilatation, in stenosis of the pylorus or when the motor activity of the stomach is impaired. After the duration of digestion has been determined, it will be well to study the contents during digestion, and the best time for this purpose is about one and one-half hours before the stomach is empty, a time at which, you will remember, the degree of acidity is at its maximum. We filter the fluid which has been removed and proceed to examine as follows:

The reaction is first determined with litmus paper, and we find it acid. An acid reaction may depend upon the presence of hydrochloric acid, or organic acids. I will first make the various tests for hydrochloric acid, of which there are many. The first, to which I will call your attention, is, that with *methyl-violet*. Two test tubes are filled with an aqueous solution of methyl-violet, the solution

being reddish-violet. To the one is added some of the gastric fluid, the other being used for comparison; should hydrochloric acid be present, the solution becomes distinctly blue. Organic acids will also render the solution blue, when in sufficient concentration, which, however, does not occur in the stomach. This is one of the oldest tests, and is very uncertain, as no reaction will occur in the presence of peptones or phosphates, and on the other hand, the presence of sodium chloride may yield the same reaction as that of hydrochloric acid. The second test is that with a solution of *tropeolin*, a few drops of which, when mixed with the gastric solution on a porcelain plate and heated, will yield a violet-red color. Another test is that with a solution of *congo red* in water, which turns blue in the presence of the acid. Filtering paper saturated with the congo solution serves as a ready test for the acid. A useless test is that with a solution of fuchsine, which becomes decolorized in the presence of the acid. The most certain and simple test for hydrochloric acid is that of *Günzberg*. Albuminoids and organic acids will not interfere with the reaction, and the smallest quantity of the acid may be determined. The *phloroglucin-vanillin* solution, which is the fluid used by Günzberg, is composed of 2 parts of phloroglucin and 1 part of vanillin to 30 parts of alcohol; a few drops of this when mixed with a similar quantity of the gastric solution and heated on a porcelain plate, will yield a red precipitate in the presence of the hydrochloric acid. From the intensity of the red color, we can determine approximately the quantity of acid present, the intensity of the redness being in direct proportion to the amount of acid.

We next determine the presence of the *organic acids*, and for all practical purposes it will suffice, if we test for lactic, butyric and acetic acids. A solution composed of 3 drops of liquor ferri perchloridi, with the same quantity of carbolic acid, in 20 cubic centimeters of water, shows the presence of lactic acid by becoming yellow; but if free hydrochloric acid is present the solution becomes colorless. Butyric acid can be recognized by its odor, that of rancid butter, and will also turn the latter solution yellow. Acetic acid is also determined by its odor, and when boiled in a test tube with the chloride of iron carbolic acid solution, will turn the latter a brownish color. The quantitive determination of hydrochloric acid* is of importance, and consists essentially of adding a $\frac{1}{10}$ normal sodium hydrate solution to the gastric secretion, 1 c. cm. of the former neutralizing 0.00365 of hydrochloric acid. Whenever the amount of hydrochloric acid is more than 0.3 per cent., it must be considered pathological. The qualitative determination of peptone† is without diagnostic

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* Let us suppose that 5.6 ccm. of soda solution was necessary to neutralize 10 ccm. of the filtered gastric fluid, then the following calculation will give the percentage of hydrochloric acid: 1 ccm. of the soda solution will neutralize .00365 of hydrochloric acid; now if 5.6 ccm. of soda solution was necessary to neutralize 10 ccm. of gastric fluid, then 56 ccm. would be necessary to neutralize 100 ccm. $56 \times .00365 = .204\%$ of hydrochloric acid.

† *Reaction for peptone.* A little of the gastric fluid is rendered strongly alkaline, and a dilute solution of cupric sulphate is added drop by drop; *a red color.*

importance, and the same may be said of pepsine,* which is never absent when free hydrochloric acid is present.

What conclusions may we arrive at after making these tests? We find hydrochloric acid absent in atrophy and amyloid degeneration of the gastric mucous membrane, which affections are rare, and also in fevers. We find it absent in the majority of cases of dilatation of the stomach due to carcinoma of the pyloric orifice, and in a few instances of so-called nervous dyspepsia. The absence of hydrochloric acid when dilatation of the stomach exists, is almost an absolute sign of carcinoma, although the normal acid reaction, and even hyperacidity, have been found in a few isolated cases, attended by dilatation of the stomach. Lactic acid is normally found in the stomach, but not longer than one hour after eating. It originates from grape sugar, by the action of microörganisms, the sugar as you know being a product of the starch from amylolytic digestion. Now, when lactic acid is found in the stomach at a longer interval than one hour after eating, it indicates abnormal fermentation, and in the majority of cases is present when hydrochloric acid is deficient or absent. Hyperacidity of the gastric secretion often occurs in ulcer of the stomach, and in certain forms of nervous dyspepsia.

The determination of the motor activity of the stomach, by means of salol, calculated by the time consumed in the appearance in the urine of salicylic acid, or the recent method of Klemperer, by measuring the quantity of its contents discharged into the intestine in a given time, are superfluous. For the practical physician, the motor activity is best determined by washing out the stomach seven hours after eating, when, if nothing is found, it indicates the sufficiency of the muscular tissue.

Let me, in conclusion, refer to a method of determining the absorptive power of the stomach. You direct the patient to swallow, on an empty stomach, a gelatine capsule, containing 0.1 gm. of iodide of potassium. You then have him expectorate on a piece of starched paper. The addition of a few drops of fuming nitric acid, if iodine is present, will give a red color, and if the iodine be in excess, a blue color. In normal cases it has been found that the absorption is accomplished in from eight to fifteen minutes. In chronic catarrh, absorption was found to be delayed for thirty minutes, and in carcinoma for three to four hours.

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* *The digesting capacity of the gastric fluid* is tested as follows: To two test tubes containing the fluid, a bit of washed fibrin is added, and to one of the tubes a few drops of 1% hydrochloric acid, and both tubes are placed in a water bath at body temperature, if after 6-12 hours the fibrin in neither tube is dissolved, *pepsine* is deficient; if the fibrin in tube containing the acid is alone digested, then the gastric fluid contains pepsine, but no *hydrochloric acid.* Normally the fibrin in both tubes should disappear in 1-2 hours.

DIAGNOSIS OF DISEASES OF THE STOMACH.

Acute Gastric Catarrh (*symptoms of dyspepsia*).—Nausea, no appetite (*anorexia*) coated tongue, bilious vomiting, slight epigastric pain, little fever, thirst, vertigo in the morning, headache, etc.

Examination of vomit shows deficient hydrochloric acid; lactic acid and mucus in excess.

Chronic Gastritis. — Early morning vomiting of glairy mucus and exaggeration of the symptom complex of the previous disease. Atrophy of the mucous membrane of the stomach and dilatation of the organ are sequelæ of the affection. In *atrophy*, when the contents of the stomach are removed, *hydrochloric acid*, or mucus, is not present.

Gastric Ulcer. — Frequent in women (anæmia). Symptoms of dyspepsia. *Fixed pain* (varying in intensity according to position of patient). Vomiting after taking of food. Hæmatemesis in about ½ the cases. An excess of hydrochloric acid is almost constant.

Gastric Cancer.—Occurs in advanced life. Symptoms of dyspepsia; emaciation and cachexia; pain is severe and independent of the introduction of food; *vomiting* occurs immediately after eating if the cancer is at the *cardiac* orifice; if at the *pylorus*, it occurs several hours after eating. Hæmatemesis in about ½ the cases. *Tumor* in about 80% of the cases. Hydrochloric acid usually absent.

Duration of disease; 1 to 5 years.

Dilated Stomach.—Symptoms of dyspepsia. *Habitual vomiting* of enormous masses containing a number of parasites (sarcina ventriculi, bacteria, etc.)

Lower border of the stomach reaches below the umbilicus. Constipation, skin dry, emaciation and diminished secretion of urine.

Cause of dilatation must be determined; may be due to *stricture of the pylorus (ulcer cicatrix or carcinoma)* or atony of the stomach.

Nervous Dyspepsia.—Pronounced dyspeptic symptoms, notwithstanding physical examination of the stomach and its contents show nothing abnormal. Nervous symptoms in other parts of the body.

VOMITING.

Vomiting consists in contractions of the abdominal muscles and diaphragm while the pylorus is closed and the cardiac orifice is open.

The *vomit center* in the medulla is irritated directly (*central vomiting*) or indirectly (*reflex vomiting*) by means of the sensory fibres of the vagus. Children vomit more easily than adults.

Forms of Vomiting: 1. Reflex from the stomach, occurs in nearly all diseases of this organ, from the action of poison, emetics, etc. 2. Reflex from the abdominal viscera; female sexual organs, peritonitis and affections of the kidneys.

Under this head may be mentioned throat irritation and vomiting after paroxysms of coughing.

3. *Central Vomiting*: Diseases of the brain and meninges, uræmia and at the beginning of infectious diseases (pneumonia, scarlatina, variola, etc.).

Vomiting is accompanied with nausea, pallor, increased pulse frequency and sweating. *Spontaneous Vomiting* not preceded by nausea is characteristic of some brain affection.

In *uræmia* the vomit has an ammoniacal odor. *Periodical vomiting* occurs as the *gastric crises* in affections of the spinal cord notably *tabes*. Vomiting before breakfast (*vomitus matutinus*) is an early symptom of potation, the vomit consisting of salvia unconsciously swallowed during the night. *Severe and persistent vomiting* unaccompanied by diseases of the stomach or abdominal viscera may be caused by infectious or brain diseases, uræmia or hysteria.

Examination of Vomited Matter.—1. Quantity. 2. Macroscopical appearance. 3. Microscopical appearance. 4. Odor. 5. Reaction.

1. *Quantity.* Large quantities in *dilatation of the stomach.* When vomiting is repeated the quantity expelled is less with each succeeding act.

2. *Macroscopical appearance.* Watery, Mucus, Bloody, Purulent, Biliary and Fæcal.

Watery vomit is represented by the *vomitus matutinus* (*morning sickness of drunkards*), consisting of swallowed saliva. The latter is recognized by the presence of *ferro-cyanide of potash* (blood red color on the addition of chloride of iron).

Watery vomit may occur in nervous dyspepsia. If the vomit contains hydrochloric acid, it is the *gastric juice* and may indicate hyper-secretion of this fluid. In Asiatic cholera the vomit is alkaline and resembles rice water.

Mucus in the vomit is observed in *gastric catarrh.*

Bloody vomit (hæmatemesis). Examination of the nose, throat and lungs must first be made to exclude hæmorrhage from these points. Bloody vomit occurs in ulcer and carcinoma of the stomach, cirrhosis of the liver, lesions of the stomach, hæmorrhagic diathesis, etc., etc. In *ulcer of the stomach* the quantity of blood is large, in *carcinoma* it has a *coffee ground* appearance, because the hæmorrhage is slow and is a long time in contact with the acid of the stomach. In hæmorrhage from the stomach, blood may pass by the bowels (*tarry stools*). *Vicarious hæmorrhage* occurs in delayed menstruation.

Fig. 12.

Fig. 12. Microscopical examination of the vomit. a. Muscular fibres b. Leucocytes. c c c' Pavement epithelium. c", Cylindrical epithelium. d. Amyloid bodies altered by digestion. e. Fat globules. f. Sarcinæ ventriculi. g. Yeast fungi. h. Comma-bacilli. i. Various micro-organisms. j. Vegetable cells. k. Fat needles.

Differential Diagnosis of Hæmoptysis and Hæmatemesis.—*Hæmoptysis.* Blood is red, frothy, alkaline and salty to the taste,

variable in amount, brought up by coughing, and there are usually physical signs of thoracic disease.

Hæmatemesis. Blood is dark, clotted and acid, large in amount, brought up by vomiting, and there are signs of abdominal disease.

Purulent Vomit is rare and is observed in phlegmonous gastritis, and from an abscess of neighboring structures perforating the stomach.

Biliary Vomit is frequent and of no special diagnostic importance. Fluid gives reactions for bile.

Fæcal Vomit usually indicates some mechanical obstruction of the intestines, and is a grave sign.

3. *Microscopical Examination.*—The elements usually recognized may be seen in Fig. 12.

4. *Odor. Fatty acids* give an acrid odor. In *poisoning* certain substances impart a characteristic odor. In *uræmia*, ammoniacal odor (conversion of urea into carbonate of ammonia.)

5. *Reaction.* Usually acid (hydrochloric or organic acids.)

The reaction is *alkaline* in *uræmia* and when large quantities of blood are vomited. Vomit of *œsophageal origin* (stricture) is alkaline.

EXAMINATION OF THE INTESTINES.

Inspection.—*Peristaltic movements* are only exceptionally seen in health, when the abdominal walls are thin and flaccid.

The movements are pathologically increased in *stenosis* of the *intestines.* According to the localization of increased peristaltic action the seat of the obstruction is determined.

In *intestinal obstruction* there is obstinate constipation, pain and tenderness of abdomen, followed by violent peristaltic action and stercoraceous vomiting with collapse.

The obstruction may be caused by; *fæcal accumulation;* history of constipation and obstruction comes on slowly; *peritoneal adhesions;* history of preceding peritonitis; *hernia, twisting of intestinal loops (volvulus)*; obstruction is sud len and occurs in perfect health. *Site of obstruction. Lower part of colon*; diffused prominence of the abdomen, fæcal vomiting, no increase of *indican* in the urine and no decided decrease of urinary secretion; *duodenum* or *jejunum;* abdomen is distended in the epigastric region while in other parts it is retracted; collapse, anuria and indicanuria (*see urine*); *ileum*

and cæcum; distension is limited to the middle of the abdomen; fæcal vomiting, collapse and anuria.

Diffused prominence of the abdomen may be caused by fat, inflation of the intestines with gas (*meteorismus intestinalis*), air in the peritoneal cavity (*meteorismus peritonei*) the result of stomach or intestinal perforation and fluid in the peritoneal cavity (*ascites*). *Retraction of the abdomen* occurs in emaciation and when an active contraction of the intestines is present, as in basilar meningitis and lead colic.

Palpation.—*Pain on pressure* may be diffused (in peritonitis and enteritis) or localized.

Localized pain in the right inguinal region may be caused by intestinal tuberculosis, typhoid fever, typhlitis or affections of the *vermiform appendix.* The situation of the latter may be determined when the patient is lying down by drawing a line from the ant. sup. spine of the ilium to the umbilicus; about 2 inches from the spine of ilium in the course of this line is the vermiform appendix.

Pain in the left inguinal region may be caused by inflammation of the descending colon (dysentery).

Intestinal tumors may be confounded with *fæcal impaction. Fæcal tumors* are usually soft and may be moulded by the fingers; they disappear after brisk purgation. If the tumor involves the *duodenum* the symptoms are similar to cancer of the pylorus (gastralgia, vomiting, dyspepsia, dilatation of stomach and retraction of abdomen). Contractions of the abdominal muscles must not be confounded with intestinal tumors. When the contractions of the abdominal muscles are irregular and associated with tympanitic distension we have the so-called *phantom tumors.* These fictitious tumors disappear when an anæsthetic is used.

Palpation of the Rectum is often necessary in the diagnosis of intestinal disease. The method of examination consists not only in the introduction of the finger, but, under the influence of an anæsthetic, of the whole hand. The hand, if small, may in many cases be introduced as far as the sigmoid flexure, and in this way the whole of the lower part of the abdomen may be explored. In order to test the calibre of the large intestine, flexible sounds, supplemented by the injection of water, constitute important diagnostic measures.

Percussion.—Over intestines containing gas, percussion yields a tympanitic sound, the height of which is influenced by the lumen of the intestines and the tension of its walls. If the intestines contain fæces in sufficient quantity, the sound while retaining the tympanitic quality, becomes duller. To determine the

relative position of the *descending colon* to other organs, it may be inflated with air by means of a sound connected with a Davidson syringe.

Auscultation of the intestines may be of importance in detecting the *gurgling* in the ileo-cæcal region and *friction fremitus* in inflammation of the serous coat of the intestines.

EXAMINATION OF THE PERITONEUM.

Free fluid in the peritoneal cavity (Ascites) is distinctly a *transudation*, and consequently non-inflammatory in origin. In inflammatory *exudations* of the peritoneum, the fluid is usually encysted and immovable.

Physical signs of Ascites. 1. *Distension of the Abdomen*, in the recumbent position it is most manifest laterally. The fluid being movable it alters its position according to the position of the patient, thus producing, in different postures, distention of definite regions. 2. The skin, in consequence of increased tension, is smooth and glistening, and the cutaneous veins are dilated in high grades of ascites (collateral routes for the intra-abdominal veins which are compressed by the fluid). 3. *Palpation* practiced in the following manner will detect *fluctuation:* put one hand on the abdomen and with the fingers of the other hand practice percussion, when, if fluid is not under too great pressure, a distinct wave is felt by the hand. This symptom may be counterfeited by an excess of subcutaneous or omental fat. If the fluid is encysted fluctuation is indistinct. 4. *Mensuration* shows an increase in the abdominal circumference. This sign is only necessary when we desire to determine during treatment the increase or diminution in the amount of fluid present. 5. *Percussion* shows a horizontal boundary line of dullness which changes position according to the position of the patient. When the patient is in the recumbent position a tympanitic sound is obtained on the anterior surface of the abdomen (*the intestines floating on the surface of the fluid,*) whereas, laterally and behind, a *dull sound* is obtained. In percussing in various positions remember that a certain time is necessary for the fluid to change its level. If the fluid is very large in amount or when the mesentery is too short, the intestines do not reach the surface of the fluid and there is dullness everywhere on percussion. When the fluid is small in amount, it first accumulates in the pelvis, in this case, put the patient on his side, so that the pelvis becomes higher and dullness will appear in the lateral region of the abdomen on the side in which the patient lies. To detect the presence of a small quantity of fluid, *i. e.*, less than 2 pints, the patient is placed in the knee-elbow position, when dullness will supersede the tympanitic sound in the umbilical region.

DIFFERENTIAL DIAGNOSIS OF ASCITES AND CYSTS OF THE OVARY.

	ASCITES.	OVARIAN CYSTS.
Form of the abdomen.	Anterior surface of abdomen flat, sides distended.	Usually distension of the anterior surface & more marked on one side.
Fluctuation.	May be felt about the dullness.	Confined strictly to the seat of dullness.
Umbilicus.	Disappears or is prominent.	Pushed upwards.
Percussion in the recumbent position.	Tympanitic sound on the anterior surface of abdomen; laterally and below dullness. Dullness also changes on position.	The opposite. Dullness does not change according to patient's position.
Examination per vaginam.	Position of uterus usually not influenced, if so, is slightly lower than normal. It is easily movable.	Uterus dislocated upwards or laterally. Mobility slight.
Microscopical and chemical examination of the aspirated fluid.	Pavement epithelium. *Specific gravity of fluid 1010–1015. Fluid clear & thin. *Paralbumen* (rare); Test: dilute fluid with water and introduce into same carbonic acid; a precipitate. Albumen small in quantity.	Columnar epithelium. Specific gravity 1018–1024. Fluid cloudy and thick. Paralbumen frequently present.

* *Transudations and exudations.* Serous exudations (inflammatory), have a greater specific gravity than transudations. When the specific gravity of a fluid exceeds 1018, it is of *inflammatory origin.* It is only a *transudation* when its specific gravity in *ascites* is less than 1012. The specific gravity is dependent upon the amount of albumen contained in these fluids and from the specific gravity, amount of albumen may be approximately determined after the formula of Reuss;

$E = \frac{3}{4}(s - 1000) - 2.8$, in which E denotes amount per cent. of albumen sought and S the specific gravity; thus fluid of a specific gravity of 1018 contains 3.95 % of albumen. These rules hold good for serous exudations only. In taking the specific gravity the fluid must be the same as surrounding temperature; for every increase of 3° C. (5.4 °F.), the specific gravity is diminished by 1 degree.

Causes of Ascites: *Ascites* without œdema in other parts of the body is usually caused by *venous stasis in the portal system*, a chronic affection of the peritoneum or *tuberculous or carcinomatous peritonitis.* In ascites due to disturbances in the portal circulation no albuminuria exists; as a rule, ascites co-existing with œdema of the extremities and cyanosis is usually caused by diseases of the heart or lungs. A chemical and microscopical examination of the urine will furnish evidence, if a nephritis is the cause of the ascites.

Physical signs of free gas in the peritoneal cavity: distension of the abdomen, dislocation of the thoracic viscera, tympanitic sound all over the abdomen, absence of hepatic and splenic dullness.

Physical signs of gas and fluid in the peritoneal cavity: tympanitic sound over gas; and dullness over fluid. Change of position alters percussion sound, the gas always being above the fluid. If the patient is shaken, a *splashing sound* is heard.

Subphrenic peritonitis (*subphrenic abscess*), is an encysted peritoneal exudation (pus) under the diaphragm, caused usually by ulcer of the stomach or affections of the intestines. If the abscess is punctured the fluid flows more rapidly during inspiration, whereas, if the fluid is confined to the pleural cavity, the flow during inspiration is less rapid because the pressure in the latter cavity sinks during this act.

EXAMINATION OF THE FÆCES.

Fæces usually consist of the residue from the process of digestion mixed with the secretions of the intestines.

Microscopical Examination.—In the microscopic examination of the stool, an inspected part is taken up with the forceps and deposited on a slide to which is added a drop or more of water. A thin watery solution of *eosin* is useful when staining is desirable.

In almost every stool we find the following (*See* Fig. 13):

1. *Vegetable Food.* Cellulose is very indigestible. It forms the frame of the vegetable form-elements and is recognized by its characteristic shape. Young vegetables are easily digested.

2. *Fat.* Present even in normal stools. Pathologically it is increased when fat resorption in the intestines is prevented and is present in icterus and diseases of the pancreas. The fat is in the form of globules or needles

3. *Muscular Fibres.* Present even in health. Increased

in digestive disturbances. Recognized by transvere striation of the fibres.

4. *Granular Detritus.* Present in every stool and indicates thorough digestion.

5. *Blood Corpuscles.* Undergo rapid destruction in the intestines. They are only recognized when the blood originates from the lower part of the large intestine and is rapidly discharged. Altered blood is recognized by Teichmann's test (*see blood*) conducted with the dried fæces.

Fig. 13.

Fig. 13. Microscopy of the stool. 1. Vegetable food. 2. Muscular fibres. 3. Fat. 4. Crystal-needles of fat. 5. Granular detritus. 6. Blood corpuscles. 7. Mucus. 8. Triple phosphates.

7. *Mucus.* Pathologically very much increased and appears microscopically as white globules imbedded in a gelatinous substance.

8. *Epithelium.* In health only sparingly present. Appears as the cylindrical variety in acute enteritis with catarrh and in Asiatic cholera.

9. *Crystals.* The triple phosphates are normally present; neutral calcium phosphate, cholesterin, leucin and tyrosin crystals may be found.

10. *Vegetable Parasites.* Bacterium termo most frequent. Sarcinæ ventriculi when they are present in the stomach. Yeast cells.

11. *Pathogenic Organisms.* The bacilli of *cholera, typhoid fever and tuberculosis.*

Cholera bacillus (comma bacillus). This is pathognomonic of Asiatic cholera. It resembles a comma and is about one-half to one-third the size of the tubercle bacillus, but plumper and thicker. It cannot with absolute certainty be recognized by microscopical examination so that the diagnosis must be based on the culture of the organism.

Method of Beijwid.—The following is applicable for the physician. A two per cent. sterilized solution of peptone is rendered alkaline by the addition of a 0.5 per cent. solution of sodium chloride and bicarbonate of sodium. This solution is then inoculated with the suspicious fæcal matter and allowed to remain in a thermostat for 24 hours. With the mixture thus obtained an addition of hydrochloric, sulphuric or oxalic acid, will yield a beautiful *violet-red color* if cholera bacilli are present. A similar reaction may be obtained with other bacilli, but it is less pronounced and requires a longer interval of time for development.

Method of Schottelius. The suspected stool is mixed with an equal quantity of alkaline meat broth and placed in an open glass for 12 hours at a temperature of 30° – 40° C. The bacilli rapidly develop on the surface, and are then examined under the microscope after staining.

Bacilli of typhoid fever. They are constantly present in this disease in the affected portion of the intestine, mesenteric glands, liver, spleen, kidneys and blood, as well as in the stool. The bacilli are straight rounded at their ends and thick (thickness equals about $\frac{1}{3}$ their length). The cover glasses are stained with methyl-blue in the usual way (*see methods of demonstrating bacteria*). Their presence microscopically in the fæces cannot with certainty be determined owing to the large number of other micro-organisms present.

Bacilli of tuberculosis. Present in the stool in intestinal tuberculosis. Their presence in the stool of phthisical persons may be due to swallowed sputum. For their demonstration (*see sputum*).

MACROSCOPICAL EXAMINATION OF THE FÆCES.

NORMAL CONDITIONS.	ABNORMAL CONDITIONS.
The *reaction* is alkaline.	*Acid* reaction in infants, and in acid fermentation of the intestines.
The *normal color* varies from a light to a dark brown and is colored by the bile. In an exclusive meat diet the color is *brownish black.* In a diet of starchy food, yellow brown; milk diet, yellow white.	Absence of gall in the intestines (icterus) stool gray, greasy and clayey. Iron and bismuth make stool black; mercurial preparations, greenish brown; santonin and rhubarb, yellow brown; logwood preparations, reddish brown. Blood from the intestines high up, tarry; from the lower parts, red.
The *quantity* in 24 hours in an healthy adult is from 100-200 grams, consisting of 75% water and 25% solids. It is dependent on the quantity and quality of the food ingested. The *consistency* is firm or thick fluid.	The *quantity* is increased in diarrhœa, and after chronic constipation, and administration of purgatives. *Consistency*, thin fluid and watery in diarrhœa.
No departure from *normal odor* is diagnostic.	The *odor* is very intense when bile is prevented from reaching the intestines (Icterus). The odor is also intense in carcinomatous and syphilitic intestinal ulcerations. When urine is present, ammoniacal odor.

The stools in *typhoid fever* have the appearance of cooked pea soup; the smell is offensive and characteristic. In *dysentery* the stools contain bloody mucus; in *cholera*, they resemble rice water. In *pancreatic disease*, the stools are colorless and contain much fat either free or as an oily scum forming tallow-like masses on cooling. In increased peristaltic action of the intestines, the stool may consist of undigested food (*lientery*).

Mucous and membranous casts may be present in membranous colitis. Portions of bowel may be found in intussusception and

exfoliated mucous membrane in dysentery and ulcerative colitis. The *chemical examination* of the fæces is without special clinical value.

EXAMINATION OF THE LIVER.

Methods. Inspection, Palpation, Percussion, Auscultation.

Anatomy of the Liver.—It is covered by the peritoneum and situated in the right hypochondriac region extending across the epigastrium into the left hypochondrium. It is the largest gland in the body, weighing from 3 to 4 pounds. It measures, in its transverse diameter, from 10 to 12 inches; from 6 to 7 inches in its antero-posterior diameter; and is about 3 inches thick at its thickest part. About ¾ of the liver is situated to the right and ¼ to the left of the median line. The upper part of the liver on the right side is covered by lung and rises under the diaphragm to the 4th intercostal space. The lower border is situated in the *scapular and middle axillary lines* at the eleventh rib; in the *mammary line* at the curvature of the ribs and in the *median line* between the xiphoid process and the umbilicus; it then takes a curved direction, attaining a point near the apex beat, between the left mammary and parasternal lines.

The gall bladder lies between the *right mammary line* and the outer border of the rectus abdominis muscle, directly under the curvature of the ribs.

Inspection.—The region of the liver in health shows no prominence unless in children who possess a relatively large liver, the latter being in a condition of physiological fatty infiltration. When the liver is enlarged and the ribs elastic (in children and young women) the hepatic region becomes prominent.

The *intercostal spaces* are preserved in hepatic enlargement and this fact serves as a means of diagnosis from pleural exudations of the right side, which cause a disappearance of these spaces. *The lower border of the liver* is rarely seen in enlargement of this organ, but the abdomen is often marked by a *furrow* which represents in many cases the lower border of the enlarged viscus. *The liver descends* during inspiration and so does the spleen but not to the same extent as the former. This respiratory dislocation serves as a clue to diagnosis in differentiating enlargement of the liver or spleen from tumors of the kidneys, stomach, pancreas, omentum and intestines, which undergo no dislocation during respiration unless adhesions exist between them and the liver and spleen, in which case the movements of these organs are communicated.

The lower liver border may be discernible irrespective of enlargement of this organ when it is dislocated by pleural exudations, tumors, deformities of the thorax and emphysema. The liver is also dislocated (in women who have borne many children) when the suspensory ligament is relaxed leading to the condition known as *wandering liver*. *An enlarged gall bladder* may likewise cause a prominence of the hepatic region. *Pulsations in hepatic region* may be transmitted from the underlying aorta in which case they are usually confined to the left lobe of the liver and the motion is an up and down one. True pulsation of the liver is an important symptom of *tricuspid insufficiency*.

Palpation.—This is the most important and certain method of examination. To accomplish palpation properly observe the following:

Place patient on his back and relax abdominal muscles by bending the legs and introducing a cushion under the shoulders. Palpate with warm hands and direct patient to open the mouth wide and breathe naturally. The palpation of the lower border is facilitated by asking the patient to take a deep breath. Fluid in the peritoneal cavity interferes with palpation, and before this can be accomplished the fluid must be aspirated or the patient put in the knee-elbow position.

In the healthy adult the surface and lower border of the liver cannot be felt. In children, however, the contrary is the case. In healthy women the lower liver border may occasionally be felt. This constitutes the *liver of tight lacing* (schnürleber) and usually involves the right lobe, which may be separated from the rest of the liver by a horizontal furrow.

In palpation of the diseased liver the following are determined: Pain on pressure, size, form, consistency and surface of the liver. These conditions will receive consideration in the appended table of liver diseases.

The gall bladder may be felt in cases of extreme emaciation. When distended with bile (*hydrops vesicæ fellæ*) and gall stones it may be palpated.

The latter condition gives the same impression on palpation as does a bag filled with stones. When distension of the gall bladder is produced by the accumulation of bile, it may be diminished in size by compression or faradization of the region occupied by the gall bladder, the effect secured being the emptying of its contents into the duodenum.

Percussion.—Two forms of liver dullness are differentiated; the *superficial or absolute* and the *deep or relative*. The *superficial or absolute liver dullness* represents the liver entirely uncovered by lung tissue. The percussion

sound over this area is flat and light percussion must invariably be employed. The *upper border* of this dullness corresponds with the lower border of the lung and is as follows:

In the right sternal line, at the lower border of the 5th costal cartilage.

In the right parasternal line, at the upper border of the 6th costal cartilage.

In the right mammary line, at the lower border of the 6th rib.

In the right axillary line, at the upper border of the 7th rib.

In the right scapular line, at the 9th rib.

Alongside of the vertebral column, at the 11th rib.

Lower border of the absolute dullness:

In the median line, between the xiphoid process and umbilicus.

In the right mammary line, at the curvature of the ribs.

In the axillary line, between the 10th and 11th ribs. Alongside the vertebral column it cannot be determined owing to the situation of the kidney.

Deep or Relative Liver Dullness—This represents not only the liver uncovered but also partially covered by lung tissue. It does not reproduce the actual size, because it is impossible for the percussion blow to reach the upper part of the liver which is covered by lung tissue of too great thickness.

Strong percussion is necessary in obtaining this form of dullness, the upper border of which runs parallel with the superficial dullness, although about 1½ inches higher; whereas the lower border corresponds with the lower border of absolute dullness. The latter form of dullness is usually relied on.

Area of Liver Dullness in Respiration. During deep inspiration the liver dullness is diminished in area owing to the descent of the lung border, about 1½ inches. When the patient lies on the left side the dullness of the liver disappears, because the right lung almost completely fills the complemental space.

In disease the liver dullness may be *absent*, *increased*, *diminished* or *dislocated*.

Liver dullness is absent in wandering liver, gas in the peritoneal cavity and in transposition of the viscera.

Increased liver dullness does not always correspond with an increase in the size of the organ, inasmuch as the dullness is influenced by the position of the lower lung border. When the lower lung border is in its normal situation and the liver border is lower than normal, sufficient evidence is furnished of an enlargement of the liver. In such a case the left lobe of the liver (*it extends normally from 7 to 8 cm. to the left of the median line*) may extend to the anterior border of the spleen.

PHYSICAL DIAGNOSIS OF LIVER DISEASES (*After Leube*).

Size of the Liver.

Diminished.	*Increased.*
Simple atrophy.	Abscess of the liver.
Atrophic nutmeg liver	Liver of Diabetes.
Cirrhosis.	Active hyperæmia.
Atrophic form (rare) of liver syphilis.	Icterus liver.
Acute yellow atrophy of the liver.	Fatty liver.
	Passive hyperæmia.
	Liver syphilis.
	Cirrhosis (*1st stage*).
	Leucæmia.
	Connective tissue—hyperplasia.
	Amyloid.
	Carcinoma.
	Echinococcus hepatis

Remark: The diseases are so arranged in the columns, that the symptoms of each are more frequent the further down the disease appears in the columns.

Consistency of the Liver.

Soft and fluctuating.	*Compact; harder than normal.*	*Hard.*
Fatty liver.	Simple atrophy.	Cirrhosis Liver syphilis.
Liver abscess	Icterus liver.	Connective tissue—hyperplasia.
Echinococ'us	Hyperæmia.	Echinococcus multiloc ularis (*becomes soft*)
		Amyloid
		Carcinoma.

Liver Border.

Smooth & sharp.	*Thick and rounded.*	*Nodular and lobulated.*
Fatty liver.	Fatty liver.	Cirrhosis (*rarely felt.*)
Icterus.	Hyperæmia.	Abscess.
Hyperplasia.	Amyloid.	Carcinoma.
Echinococcus.		Liver syphilis.
Simple atrophy.		

Surface of the Liver.

Smooth.	*Nodular.*
Cirrhosis(*1st stadium*).	Cirrhosis.
Hyperæmia	Abscess.
Fatty liver.	
Icterus liver	Liver syphilis.
Elephantiasis.	
Amyloid.	Carcinoma
Leucæmia.	
Diabetes.	Echinococcus.
Acute yellow atrophy.	

Icterus.

Absent.	*Rare.*	*Frequent.*
Amyloid.	(only when the biliary ducts are directly involved); in	Hyperæmia.
Pylephlebitis adhæsiva.	Echinococcus.	Echinococcus multiloc.
		Cirrhosis.
Fatty liver.	Liver syphilis.	Carcinoma
		Elephantiasis.
	Abscess.	Icterus liver.

Ascites.

Absent.	*Present.*
Fatty liver.	Carcinoma.
	Liver syphilis with cicatrices
Elephantiasis.	Cirrhosis.
	Pylephlebitis adhæsiva.
Icterus—liver.	Amyloid.
Echinococcus.	Echinococcus multiloc.
Abscess.	Hyperæmia, (constant in later stages).

Pain.

Present.
Echinococcus multiloc.
Acute yellow atrophy.
Carcinoma.
Liver—syphilis.
Abscess.

Enlargement of the Spleen.

Absent.	*Present.*
Carcinoma	Echinococcus simplex (rarely after stasis in the *portal system*)
Fatty liver.	Hyperæmia.
	Echinococcus multiloc-ularis.
	Liver syphilis.
	Cirrhosis. Amyloid.
	Also in general infection caused by acute yellow atrophy and abscess.

Pleural exudations, tumors of the lung and pleura and consolidation of the lung on the right side render percussion of the upper border of the liver impossible, as one dullness cannot be separated from another. An apparent increase of liver dullness can also be produced by solid matter in the colon and stomach.

Diminished liver dullness does not always correspond with a decrease in the size of the organ. An apparent diminution in the area of dullness may occur; when the transverse colon is pushed between the liver surface and the chest wall, in emphysema of the lung and in distension of the stomach and intestines.

Dislocation of the liver dullness occurs when the liver is dislocated upwards in inflation of the intestines, ascites, tumors of the abdominal organs and contraction of the right lung. A dislocation downwards occurs in pulmonary emphysema, pleural exudations, mediastinal tumors, etc.

Auscultation of the liver is usually devoid of positive results. *Friction murmurs* in perihepatitis and *arterial murmurs* either transmitted from the heart or occurring spontaneously in the liver, may at times be heard.

EXAMINATION OF PANCREAS, OMENTUM AND RETROPERITONEAL GLANDS.

Pancreas. Tumors of this organ may occasionally be palpated in the epigastrium, directly under the lower border of the liver.

Omentum. This is only palpable immediately about the umbilicus in rare instances, when thickened by inflammation or is the seat of new growths.

Retroperitoneal Glands. They may be secondarily involved in carcinoma. They are deeply seated in the abdomen on a level with the umbilicus.

EXAMINATION OF THE SPLEEN.

Methods. Inspection, Palpation, Percussion, Auscultation.

Anatomy of the Spleen.—The spleen is a long, nearly oval shaped organ, lying in the left hypochondrium, between the 9th and 11th ribs. Its anterior end does not normally reach, or at any rate, go beyond a line drawn from the tip of the 11th rib to the left sterno-clavicular articulation (*costo-articular line*). Its posterior border usually terminates at a distance of 1 inch from the 10th dorsal vertebra. It has 3 surfaces. The *outer convex surface* is directed towards the under surface of the diaphragm, the *inner concave surface* is directed towards the fundus of the stomach, while a small part of its under surface covers the upper

part of the left kidney. The *weight* of the spleen in the male is about 7 ounces; female, 6 ounces. It is about 5 inches long, 3½ inches broad, and 1¼ inches thick. In old age the organ decreases in weight.

Inspection.—The splenic region shows no departure from the normal unless the spleen is considerably enlarged, in which case, a prominence of the left hypochondrium and the adjacent abdominal region may be noted. When the spleen enlarges it grows downwards from the left hypochondrium into the umbilical and hypogastric regions, and may, in excessive enlargement, occupy a large portion of the peritoneal cavity.

An *enlarged liver* grows downwards from the right side; an *enlarged uterus* grows upwards from the hypogastric region, while an *enlarged ovary* grows upwards from either inguinal region. Splenic tumors are nearly always recognized by their *dislocation on change of position* and *during respiration.*

Palpation.—This is the most important of all the methods of examination, and we are less likely to err in palpation than percussion. *A normal spleen is rarely felt.*

In palpation put patient in the *right diagonal position*; patient lies on the right shoulder with the left arm raised toward the head and midway between the dorsal decubitus and side position. This position is the most favorable for palpation of the spleen. The fingers of the right hand are now pressed downwards and upwards into the space between the 10th and the free end of the 11th ribs, and the patient is told at the same time to take a deep breath, when, if the organ is enlarged, it is felt as a rounded body, receding from the fingers during expiration. If the organ is soft only an increased resistance is felt. The spleen may be palpated when it is dislocated and not necessarily enlarged.

The following conditions are observed in palpation: form, size, consistency, pain, mobility and the condition of the surface of the spleen.

Form of the Spleen. Tumors of the spleen usually reproduce on a larger scale the original form of this organ. The anterior margin of the normal organ presents from one to four *notches* which are pronounced in enlargements of this organ. Palpation of these notches is of great importance in differential diagnosis.

Size of the Spleen. This varies according to the causes concerned in the enlargement. The largest spleen is usually of leucæmic origin.

Consistency of the Spleen. As a rule, the larger the

spleen the harder the consistency. Acute are usually of softer consistency than chronic splenic enlargements.

Pain. Usually absent in splenic enlargements unless the peritoneum is secondarily involved.

Mobility of the Spleen. This is effected by pressure with the hand, change of position and during respiration.

Condition of the Splenic Surface. It may be nodular and irregular; in thickening of the capsule (rare), carcinoma, sarcoma, echinococci, gummata, cysts, abscesses and varicose dilatation of the splenic veins.

Mobility of the Spleen during inspiration may be absent when the enlarged organ presses on the diaphragm and interferes with its contraction. In women the spleen may leave its normal situation (*wandering spleen*). It is recognized by its form, by the notches on its anterior margin, by excessive mobility, absence of dullness in the splenic region, and by its relation to the colon. Wandering and enlarged spleens lie in front of the colon a relationship which can be recognized by inflation of the latter. Palpation of the spleen may give rise to a cough (*spleen cough*) and is due to mechanical irritation of the peripheral branches of the pneumo-gastric nerve.

Percussion.—A large part of the spleen is covered by lung which interferes with percussion of the entire organ. Only that portion can be percussed which is not covered by lung.

The Area of Percussion Dullness is bounded above by the left lower lung border, which in the right diagonal position is at the 9th rib in the middle axillary line ; the lower border runs parallel with the 11th rib, and just before reaching the left scapular line it joins the lateral border of the left kidney. The breadth of the splenic dullness in the middle axillary line is from 2 to 2½ inches. The normal splenic dullness does not reach forward beyond the *costo-articular line.* The *percussion blow* must be very light. The splenic dullness is separated from the resonance of the lung above, and on the sides, from a tympanitic sound. The figure of dullness will alter according to the position of the patient, hence the necessity of always percussing in one position, the right diagonal being preferred. On inspiration, owing to the descent of the lower border of the left lung, the splenic dullness is diminished.

In health the figure of splenic dullness is influenced by so many conditions that percussion ceases to be an exact method of examination. The position of the left lower lung border will either increase or diminish the dullness, and a fatty omentum or accumulations in the stomach and colon, exert a decided

influence on the percussion area of the spleen. *If an apparent enlargement of the spleen is found on percussion and the organ is not palpable, always question the correctness of the percussion.* The excellent advice of Piorry should be remembered, viz. : That the certainty of splenic percussion increases after free purgation.

The splenic-dullness may be increased, diminished or absent.

Increased splenic dullness occurs in certain acute infectious diseases (typhoid, malaria, pyæmia, etc.), obstruction of the portal circulation, amyloid degeneration, leucæmia and new growths in the organ.

Enlargement is assumed to exist when the percussional breadth is increased; when the dullness goes beyond the costo-articular line (in only $\frac{1}{10}$ of the cases does the normal splenic dullness go beyond this line), and when the sense of resistance on percussion is increased. An apparent enlargement is observed in pleural exudations of the left side, and in infiltration of the lower portion of the left lung.

Diminished splenic dullness occurs in *pulmonary emphysema* owing to encroachment of the dilated lung on the area of splenic dullness, and when the intestines are distended with gas.

The splenic dullness is absent when the spleen is absent (rare), in wandering spleen, and when air is present in the peritoneal cavity ; the air occupies the region between the surface of the spleen and the thoracic wall thus substituting a tympanitic for a dull sound.

Auscultation of the spleen. When the capsule of the spleen is roughened *friction sounds* may be heard. *Arterial murmurs* have been heard in intermittent fever.

CHAPTER XI.

EXAMINATION OF THE GENITO-URINARY ORGANS.

EXAMINATION OF THE KIDNEYS.

Anatomy. The kidneys lie on both sides of the vertebral column from the level of the 12th dorsal to the 3d lumbar vertebra. The right is about ¾ of an inch lower than the left kidney. The kidney is about 4 inches long, 2 inches wide, 1 inch thick, and weighs in the male about 5¼, and in the female 4¾ ounces. The right kidney encroaches above on the liver, the left kidney on the spleen. The outer border of the kidneys is about 4 inches external to the spinous processes of the vertebræ, and corresponds with the outer border of the *sacro-spinalis muscle.* The anterior surfaces of the kidneys are covered by the parietal layer of the peritoneum. The *ascending colon* lies in front of the right kidney, the *descending colon* in front of the left.

Physiology. In the kidneys the water is filtered off through the *glomeruli* with certain inorganic salts, and is dependent entirely upon blood pressure. The *secretion* proper takes place in the epithelium lining the tubules, and is practically independent of blood pressure.

Examination of the Normal Kidneys. The usual physical signs are without special value, even under the most favorable conditions (flaccid and thin abdominal walls, atrophy of the sacro-spinalis and quadratus lumborum muscles).

Inspection. When the kidney is excessively enlarged, a distension of the region occupied by it may be noted.

Palpation. This is the most important local method of examination.

In palpating the kidney the patient is placed on his back with the legs flexed on the abdomen and *bimanual palpation* is made with one hand on the lumbar region, the other on the abdomen. *Pain on pressure* is occasionally experienced in acute nephritis,

renal tumors, inflammatory hydro-nephrosis and perinephritis. Large tumors of the kidney (*sarcoma, carcinoma*) may be felt as irregular nodules; whereas in hydro-nephrosis the kidney surface is smooth, tense and fluctuation may be present. Echinococci of the kidney may be felt as elastic cysts, eliciting a peculiar vibration, similar to the sensation perceived on striking a mass of jelly (*fremitus of hydatids*). Tumors of the kidneys exhibit no movement during inspiration or by pressure, unless a wandering kidney exists.

Wandering or *floating kidney* is comparatively frequent in women who have borne children. The right kidney is the one usually involved (greater length of the renal vessels and the looser attachment of the right kidney).

An important point to remember in differential diagnosis is, that the colon always lies in front of the kidney, and inflation of the colon from the rectum with gas or air is often necessary to establish this relation.

Percussion.—Only the outer convex and lower border of the kidney can be percussed from the tympanitic sound of the neighboring intestines. Even this is only possible when the latter are empty.

Examination of the Ureters and the Bladder—Ureters.—They are frequently palpated with the hand introduced into the rectum. In women they may be felt by vaginal examination. The *endoscope* has been used to render manifest the opening of the ureters into the bladder, and to determine their length (normal, 16 to 18 inches) by the introduction of sounds. The ureters are thickened and painful on pressure in *cysto-pyelitis* and *tuberculosis of the urinary apparatus*. They may be dilated in *pyelitis calculosa*.

Bladder.—This may be palpated through the abdominal wall when it is distended with urine. When abnormally distended it may almost reach the ensiform cartilage. Pressure on the distended bladder occasions a desire to urinate. Percussion is serviceable in marking out the distended viscus. The internal examination of the bladder may be made by digital examination *per rectum*. *Auscultation* practiced with the sound moved about in the bladder is valuable when calculi are present. A filled bladder has been confounded with tumors of the pelvic viscera; the use of the catheter will solve the difficulty. The improved apparatus of Leiter and Nitze, with which the interior of the bladder is illuminated by means of the electric light is a valuable aid to diagnosis in the hands of experts.

DIFFERENTIAL DIAGNOSIS OF DIFFUSED DISEASES OF THE KIDNEYS.—(*After Leube.*)

		Passive hyperæmia.	*Acute nephritis.*	*Chronic nephritis.*			*Amyloid kidney*
				Chronic nephritis (large white kidney)	*Secondary contracted kidney*	*Slowly developing chronic nephritis, primary (contracted kidney)*	
ETIOLOGY		Diseases of the heart and lungs; thrombosis of the renal vein or inf. vena cava.	Catching cold, acute poisoning (cantharides) acute infection (scarlatina, pneumonia, typhus, diphtheria, sepsis, etc.).	Acute nephritis (scarlatina, etc.), slowly acting cold (moist habitations), malaria, valvular cardiac lesions and phthisis.		Gout, saturnismus, potus, pyelitis (lithiasis), syphilis.	Suppuration, especially caries, phthisis pulm. Lues (particularly amyloid contracted kidney). Malaria carcinoma, ulcers of the foot, etc., (seldom.)
Condition of the Urine.	Quantity.	Small.	Small.	A little less than normal, about 1 liter.	Abundant or at least normal.	Very abundant.	About normal; varies.
	Color.	Dark red.	Pale to dark red; cloudy.	Meat-water color, cloudy.	Comparatively light.	Light and pale.	Pale yellow, light.
	Specific gravity.	High.	High.	A little higher or normal.	Slightly less than normal.	Low.	Normal or diminished.
	Blood.	Absent (a few red corpuscles may be present).	Abundant.	Usual.	Usually a little.	Red corpuscles usually absent.	Absent.
	Albumen.	Moderate though varying in quantity.	Abundant.	Abundant.	Moderately plentiful.	Slight.	Absent at times; as a rule plentiful.

DIFFERENTIAL DIAGNOSIS OF DIFFUSED DISEASES OF THE KIDNEYS.—*Continued.*

		Passive hyperæmia.	*Acute nephritis.*	*Chronic nephritis.*	*Secondary contracted kidney.*	*Primary contracted kidney.*	*Amyloid kidney.*
Condition of the Urine.	Sediment.	A moderate quantity of uric acid salts, hyaline cylinders and red blood corpuscles.	White and red blood corpuscles, blood and epithelial casts, uric acid salts.	White and red blood corp'scles; casts of all kinds; fatty casts particularly abundant.	Casts of all kinds.	Very slight, particularly hyaline casts (narrow and broad).	Absent at times, very few hyaline and fatty casts; leucocytes.
	Solid constituents.	About normal. Increased percentage of urea, although not absolute.	Excretion of urea, chlorides and phosphates, diminished.	Diminished excretion of solid constituents.	Decided diminution of solid constituents.	Decided diminution in the excretion of solid constituents.	Normal.
Hypertrophy of the heart.		Caused by the etiological factor.	Absent as a rule.	Occasionally present.	Usually present.	Almost always present.	Absent, unless an amyloid contracted kidney is present.
Hydrops.		Caused by etiological factor, usually stationary especially in the lower extremities.	Pronounced (rarely absent); site varies.	Pronounced (cavity hydrops).	Moderate (anasarca or cavity hydrops).	Usually absent, present later when heart is insufficient.	Usually very pronounced.
Uræmia.		Absent.	Frequent, especially in nephritis following colds and scarlatina.	Comparatively frequent.	Frequent.	Very frequent.	Absent, unless an amyloid contracted kidney exists.
Other symptoms.		General venous stasis (liver hyperæmia, etc.).	Symptoms of the infectious disease and poisoning.	Pronounced paleness of the skin, retinitis, bronchitis, etc.; inflammation of internal organs.			Retinitis absent, symptoms of the causal affection. (See *Etiology*).
Cause of death.		Cardiac weakness, infarctions, etc.	Uræmia or inflammation of internal organs; pulmonary œdema, pneumonia serosa.	Uræmia or more frequently from inflammation of internal organs.	Uræmia, cerebral hæmorrhage, insufficiency of the heart, inflammation of internal organs.		From the causal affection, exhaustion or emaciation.

DIAGNOSIS OF DISEASES OF THE BLADDER.

Cystitis.—In the acute form of this affection the bladder region is painful; the *urine* is diminished, cloudy and acid in reaction. The sediment is abundant, and contains leucocytes, red blood corpuscles and bladder epithelium. In *chronic cystitis*, the urine is cloudy, dirty brown in color, alkaline (ammoniacal fermentation) and contains a sediment of pus.

Cystitis Calculosa (*bladder calculi*).—Usual signs of cystitis. Hæmaturia is a frequent complication. Crystalline deposits are present in the urine, or they are expelled as gravel. Pain is often intense, and radiates towards the urethra and glans penis. The pain is usually increased by active movements of the body. Strangury. The diagnosis is only positive when a calculus is detected by means of the sound.

Cancer of the Bladder.—Hæmaturia, and pain in the bladder region uninfluenced by movement. Cancerous particles may be expelled with the urine. Enlargement of the inguinal glands and cachexia. The diagnosis is positive when a tumor can be palpated from the rectum or vagina, or by means of a catheter.

Nervous Disturbances.—In paralysis of the longitudinally arranged muscular fibres of the bladder (*Detrusor vesicæ*) there is retention of the urine (*Ischuria paralytica*); the retention continues until the pressure of the accumulated fluid overcomes the resistance of the circularly arranged muscular fibres of the bladder (*Sphincter vesicæ*) and the urine is expelled drop by drop (*Incontinentia urinæ* or *Enuresis paralytica*). In spasm of the bladder (*cystospasmus*) involving the *Sphincter muscle* the urine is passed in small quantities (*Dysuria spastica*) or when the spasm is pronounced, no urine is expelled (*Ischuria spastica*). Spasm of the *Detrusor* is characterized by a sudden and frequent desire to urinate.

URINARY CALCULI.

Calculi are solid masses formed by deposition of inorganic or organic constituents of the urine. They may occur of any size from mere granules (*gravel*) to masses as large as the fist (*calculi*). Calculi are usually composed of a *nucleus* consisting most frequently of oxalate of lime or organic matters (mucus, blood, parasites or foreign bodies).

Calculi in Acid Urine.—*Uric acid.* Hard and heavy, of a reddish yellow color. Moisten some of the powder with a drop of nitric acid and slowly evaporate in a dish. If uric acid is present, an orange-red mark is left which turns purple on being moistened with ammonia (*murexide test*). *Oxalate of Lime* (*mulberry calculi*) occur next in frequency to uric acid, forming about $\frac{1}{3}$ of all calculi. Very common in children, and may be passed as hemp seed-like bodies. They are hard, irregular and dark brown or purple in color. The powdered calculus is not dissolved by acetic acid but dissolves without effervescence in mineral acids.

Cystine Calculi (very rare); yellowish-white color with a granular, glistening crystalline surface. Fragments dissolve in ammonia and separate on evaporation as regular hexagonal crystals of cystine. *Xanthine* (very rare); yellow brown color, and glistens like wax on friction.

Calculi found in Alkaline Urine.—*Mixed phosphates* (fusible calculi); consist of the mixed calcium, ammonium and magnesium phosphates. These substances are usually constituents of other calculi which have remained in the bladder until the urine has become alkaline and ammoniacal. Under these circumstances they form a very friable crust over the calculi. They are soluble in acids and fuse under the blow-pipe into a glassy slag.

Phosphate of Lime (rare); white and chalky, break with an earthy fracture and appear regularly stratified.

Carbonate of Lime (rare); small rounded bodies and generally very hard. Dissolved with effervescence in hydrochloric acid.

The *Uro-stealith calculus* is probably composed of some fatty material. *Blood calculi* only occur in renal hæmaturia.

Analysis of Urinary Calculi.—The calculus is powdered or a portion of each layer, or any layer scraped off is powdered and burnt on a piece of platinum foil over a Bunsen flame or spirit lamp. The specimen may: 1. Carbonize and disappear. 2. May partly carbonize and leave a residue. 3. May undergo little or no blackening.

1. *If the specimen burns and leaves no residue* the calculus consists of *uric acid urate of ammonia, cystine, xanthine* or *fibrin.* A fresh portion is digested with a strong solution of ammonia

and filtered; the filtrate is evaporated depositing *crystals of cystine* (recognized microscopically). The *insoluble portion* is dissolved in *nitric acid* and the solution evaporated; a yellow residue which turns red on adding caustic potash in the cold, and violet when heated, indicates *xanthine*; a pink residue, giving a purple color with ammonia, indicates *uric acid.*

2. The *specimen carbonizes and burns, leaving a residue.* The calculus contains *organic* and *inorganic* constituents. To determine these, boil in water another portion of the powdered calculus filter while hot; a deposit on cooling indicates *urates* or *uric acid.* The insoluble portion in boiling water should be treated with *acetic acid* and filtered. Filtrate may be tested for *earthy phosphates*, whilst the residue dissolved in hydrochloric acid, and the solution super-saturated with ammonia, will give a crystalline deposit if *oxalate of lime* is present.

PATHOLOGICAL CONCREMENTS.

Fæcal calculi (enteroliths), consist of organic substances and inorganic salts (phosphate of calcium, ammonio-phosphate of magnesium, etc). They should be dissolved in muriatic acid and examined in the usual way.

Salivary calculi generally consist of carbonate of lime.

Rhinoliths are calcareous concretions formed around some impacted foreign body in the nasal cavity. They may be due to the inspissation of nasal secretions.

Gall Stones.—They consist of *cholesterine* or *bile pigment* or rarely of carbonate or phosphate of lime. Biliary concretions are usually deposited around a nucleus of inspissated mucus. *Cholesterine calculi* are smooth, light-colored faceted when multiple and quite hard. Their crushed surface shows radiating lines of fracture. Cholesterine is detected by dissolving a portion of the powdered calculus in hot alcohol and filtering; after cooling it crystallizes in slender plates. If cholesterine is dissolved in chloroform and sulphuric acid added, a cherry-red color is formed. Gall-stones consisting of *bile pigment* are almost black in color, small and irregular in shape, very friable, and occur in large numbers. *Test for bilirubin* with Gmelin's reaction.

THE URINE.

Examination of the urine is essential in learning the condition of the kidneys and bladder; and determining qualitatively and quantitatively those products (*urea*, etc.) which originate in the disintegration of albumen. The force of the heart's action and certain affections of other organs may also be determined. The

products of the decomposition of *fat* and *carbo-hydrates* pass out of the body as carbonic acid and water by the lungs; whereas the products of albumen pass out in the urine.

Normal Urine.—It is a transparent amber-yellow colored fluid; consistency of water; saline taste; aromatic odor; acid reaction; specific gravity, 1015 to 1025, at a temperature of 60° F. The quantity excreted in 24 hours in health, by men, is about 1500—2000 ccm. (40—60 ounces); and by women 1000—1500 ccm. (30—40 ounces).

Reaction.—Normal urine is acid, due to the acid phosphates of sodium ($Na\ H_2\ PO_4$). The reaction is determined by blue and red *litmus paper* (tumeric paper is of little value). Blue litmus paper will be turned to a red color, if urine is acid; if alkaline, it will turn red litmus paper blue. *Amphoteric reaction* is present when red litmus paper is turned blue and blue litmus paper red. To distinguish between *fixed alkali* and that due to the presence of ammonia (decomposed urine), the litmus paper after being dipped in the liquid is allowed to dry; the blue color disappears in ammoniacal urine, but remains if the alkalinity is due to a fixed alkali.

The degree of acidity of the urine is determined by acidimetry, *i. e.*, titration with a decinormal solution of potassium hydrate, expressing result in the corresponding amount of oxalic acid.

Alkaline Reaction from fixed Alkalies.—This occurs after a vegetable diet, the use of caustic alkalies, alkaline carbonates or alkaline salts of the vegetable acids, by mixture with blood or pus, etc. During digestion urine may become alkaline.

Alkaline Reaction from a Volatile Alkali.—This results from the presence of bacteria decomposing urea into ammonium carbonate:

$$\underset{\textit{urea}}{CO\ (N\ H_2)_2} + \underset{\textit{water}}{2\ H_2\ O} = \underset{\textit{ammonium carbonate}}{(NH_4)_2\ CO_3}$$

This alkaline fermentation may occur within or without the body.

The alkalinity from fixed alkalies causes precipitation of the earthy phosphates while ammoniacal alkalescence causes the formation of triple-phosphate.

An acid reaction in the presence of *pus* is strong evidence of its renal origin. *Chronic cystitis* is usually associated with an alkaline urine.

Color.—This may vary in health owing to concentration or dilution, or in disease owing to the presence of pathological pigments.

The urine of diabetes, hysteria, anæmia, convulsions and contracted kidney is usually pale; in fever it is high-colored owing to a diminished quantity of water.

The normal urinary coloring bodies are indican and urobilin. *Indican* (*uroxanthine*) is increased (*indicanuria*) in obstruction of the intestines (absorption of indol of the fæces), peritonitis, chronic constipation, and occasionally in health.

Test for indican: Mix with the urine an equal part of hydrochloric acid and 1 or 2 drops of a concentrated solution of calcium chloride diluted one half with water. The chloride of calcium solution is added drop by drop until a blue color appears. A few ccm. of chloroform are then added and the whole shaken, which brings out the indigo.

Urobilin colors the urine red or brownish-red. Present in health in small quantities. It is increased in febrile disease and during the resorption of large blood extravasations.

Tests for urobilin: *Spectroscope.* An absorption line between the green and the blue of the spectrum.

Chemical: Add to the urine 2—5 drops of a 10% solution of chloride of zinc and enough ammonia afterwards to re-dissolve the precipitated oxide of zinc; a green fluorescence is observed if the test-tube is held against a dark background.

Blood-coloring matter. The urine is either bright red with a greenish iridescence owing to the presence of oxy-hæmoglobin or a dark brownish-red, owing to the presence of meta-hæmoglobin.

Blood-coloring matter occurs in: 1. *Hæmaturia* (coloring matter present in combination with blood corpuscles). 2. *Hæmoglobinuria* (coloring matter is in solution without blood corpuscles in sediment). Hæmoglobinuria results from hæmoglobinæmia which is caused by *poisoning* (chlorate of potash, mineral acids, etc.), infectious diseases, burns, etc.

Bile coloring matter. The urine is of a beer-brown color and has a yellow foam on shaking.

Medicinal agents. The urine is brown or black after

the ingestion of carbolic acid and gallic acid; yellow after santonin and chrysophanic acid.

A dark brown or blackish color of the urine occurs in melanotic tumors, owing to the elimination of *uromelanin*, a black coloring body similar to the choroidal pigment.

Quantity.—1. Increased (*polyuria*) in hydræmia. 2. Contracted kidney (caused by the accompanying hypertrophy of the left ventricle increasing blood pressure). 3. During the resorption of exudations and transudations. 4. Diabetes insipidus and mellitus. 5. After great thirst (*polydipsia*).

Transitory polyuria is observed in nervous individuals after mental excitement, and after the use of coffee, beer, wine and diuretics.

The quantity is decreased: 1. From loss of water (perspiration, diarrhœa, formation of exudations and transudations). 2. Fever. 3. Diminished blood pressure (valvular heart diseases). 4. Acute and chronic parenchymatous nephritis. 5. Obstructions to the excretion of urine (strictures of the urethra and ureters).

Transparency.—Normal urine is clear. An opacity occurring in urine of an acid reaction may be due to uric acid salts (urine clears on heating) or to certain organic constituents recognizable with the microscope. An opacity in alkaline urine is usually due to the phosphates or more rarely to calcium oxalate or organic constituents. The urine is rendered opaque in chyluria owing to the presence of emulsified fat (*galacturia*) or to large fat drops on the surface of the urine (*lipuria*).

Specific Gravity.—This is ascertained with the urinometer (*aräometer*) or for greater accuracy by a specific gravity bottle (*picnometer*). In taking the specific gravity, the urine of 24 hours should be collected and a sample taken from it.

The specific gravity may vary from 1002 (contracted kidney and diabetes insipidus) to over 1060 (diabetes mellitus and fever). As a rule, a low specific gravity indicates a diminished and a high specific gravity, an increased excretion of urea.

Estimation of Total Solids.—This can be approximately determined, if the last two figures of the specific gravity be multiplied by the co-efficient 2.33 (*Hæser*) or by 2 (*Trapp*) or 2.2 (*Lœbisch*); the result, is the amount in grams in 1000 ccm. of urine. If 1200 ccm. be the amount of urine and the specific gravity 1022, then

$$22 \times 2.33 = 51.26 \text{ grams in 1000 ccm.}$$

$$\text{or in 1200 ccm.} = \frac{51.26 \times 1200}{1000} = 61.51 \text{ grams.}$$

Odor.—It is ammoniacal in decomposition of the urine. In diabetes the urine has a fruity odor. Spirits of turpentine yield an odor like violets; copaiba, cubebs, and oil of sandalwood, an aromatic odor. Urine containing *cystine* smells at first like sweetbriar, but afterwards becomes very offensive. The odor of the urine after taking garlic and asparagus is well known. The urine has a putrid odor when it contains decomposing blood or pus.

CONSTITUENTS OF NORMAL URINE.

Urea [$CO(NH_2)_2$].—The daily amount excreted by healthy persons is between 20–40 grams (300–600 grains). Urea is increased in an albuminous diet, and in an increased loss of the albumen of the body (diabetes mellitus, phosphorus poisoning, fever, etc.) It is decreased in inanition, in a deficient nitrogenous diet, in acute yellow atrophy of the liver and in diseases of the kidney.

Urea is a soluble organic salt and represents the chief ultimate product of nitrogenous waste; it constitutes about 3 per cent. by weight of the urine. It crystallizes in silky, four-sided prisms, with oblique ends or in delicate white needles.

Chemical Test (*biuret reaction*).—Heat a specimen of urea until it ceases to give off ammoniacal vapors. If *potassium hydrate* and a drop of *cupric sulphate solution* are added to the residue, the color is changed to a reddish violet.

Microscopical Test.—This is important in detecting urea in sputum, vomit, transudations, etc., in the diagnosis of uræmic conditions. Evaporate fluid to the consistency of syrup, extract with alcohol and filter. The filtrate is then evaporated, the residue dissolved in a little water and concentrated nitric acid added to it. After a little while, hexagonal crystals of nitrate of urea appear.

The quantitative determination of urea may be accomplished by the *balance, precipitation* by means of certain standard liquids

and *volumetric analysis*. The last is the only ready and rapid method suitable for the physician. It is based upon the fact that urea decomposes into nitrogen and carbonic acid in the presence of certain bodies and by ascertaining the quantity of gas produced, the urea is estimated.

The *ureometer of Doremus* is a simple and useful instrument. It consists of a bulb and graduated tube, and a small curved pipette, to hold 1 ccm. of urine. The tube is filled with *hypobromite solution* of the usual strength to the mark indicated on the long arm of the apparatus and then water is added to fill the rest of the arm and lower part of the bulb. The pipette filled with urine is then introduced as far into the bend as it will go and the nipple compressed to expel all the urine. The tube is so graduated that each of the small divisions = .001 gram of urea. The percentage can be obtained by multiplying the weight of the urea used as indicated by the graduation on the glass by 100. The simple apparatus of Squibb is sufficiently accurate for clinical purposes.

Uræmia represents a group of nervous symptoms caused by retention in the blood of the urinary excretory products (particularly urea). The mild symptoms of uræmia, are: sleepiness, headache, nausea, vomiting, dyspnœa (*uræmic asthma*) and Cheyne-Stoke's respiration; *severe symptoms:* coma, delirium, convulsions and amaurosis.

Uric Acid (C_5 H_4 N_4 O_3).—The daily amount passed is between 0.2 gram (3 grains) and 1.0 gram (15 grains). Nitrogenous food increases and carbohydrates diminish uric acid in the urine. It is also diminished during, but increased after an attack of gout. It is *increased* in affections of the respiratory (pneumonia, bronchitis, etc.) and circulatory systems. It is very much increased in *leucæmia*.

Uric acid is slightly soluble in water (1 to 18,000), and insoluble in alcohol and ether. The crystals of uric acid separated from the urine appear to the naked eye as small reddish-brown particles (*brick-dust sediment*). Microscopically (Fig. 14), they present a variety of shapes, the most frequent are the whetstone or lozenge form rounded off at their obtuse angles. Other forms resemble barrels, rosettes, combs, etc.

Test for uric acid (murexide test).—See Urinary calculi, Quantitative estimation. Acidulate 200 ccm. of urine with 10 ccm. of *hydrochloric acid* and set aside for 48 hours. The separated crystals are collected by filtering the fluid, then dried and weighed.

Hippuric acid ($C_9 H_9 NO_3$). Daily amount excreted 0.1—1.0 gram (1½—15 grains). It is increased by a vegetable, and diminished by an animal diet. It is readily soluble in alcohol. Crystallizes in colorless, long, four-sided rhombic prisms, which frequently form stellate bundles.

Oxalic acid ($C O O H)_2$. The amount excreted is about 0.02 grams (⅓—¼ grain) in 24 hours. It appears in the sediment as *calcium oxalate* in small octahedral crystals, insoluble in acetic and soluble in hydrochloric acid.

Creatinine ($C_4 H_7 N_3 O$). About 1.0 gram (15 grains) is daily excreted.

Xanthin and Sarkin. Extractives of the urine allied to uric acid in chemical composition, but of no special clinical value.

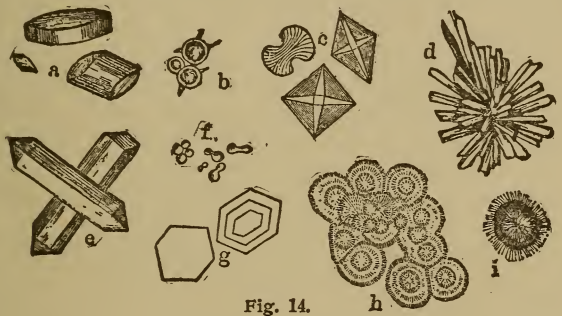

Fig. 14.

Fig. 14. a, Crystals of uric acid. b, Urate of soda. c, Calcium oxalate. d, Calcium phosphate. e, Triple phosphate. f, Calcium carbonate. g, Cystin. h, Leucin. i, Tyrosin.

Indican.—See color of the urine.

Phenols. Carbolic acid ($C_6 H_5 OH$), *hydroquinone* and *cressol* exist in the urine as the ethereal sulphates. They are increased in decomposing processes in the body.

Test. Add to 100 ccm. of urine, 5 ccm. of concentrated sulphuric acid, and distill the whole in a retort. Bromine water is then added to the distillate, and if carbolic acid is present, a yellow-white precipitate of tri-bromo-phenol is formed.

NORMAL INORGANIC CONSTITUENTS.

The Chlorides. Principally combined with sodium as common salt. The amount of sodium chloride excreted in 24 hours is about ½ the amount of urea present, *i. e.*, between 11 and 15 grams (150—225 grains).

The chlorides are *decreased* in all acute febrile diseases (especially pneumonia) and reappear with convalescence. This is particularly the case in diseases accompanied by exudations and transudations, which retain the surplus chlorides until their formation is complete.

Test. Add to the urine in a test tube a few drops of *nitric acid*, and then a *nitrate of silver* solution until no more precipitate forms. *Precipitate* is *dense and curdy*, if the chlorides are normal; *milky* if diminished; and *faint* if almost or entirely absent. The relative amount may be estimated by comparison with a normal specimen of urine.

The Phosphates. Phosphoric acid occurs in the urine, in part as sodic and potassic phosphate (*alkaline phosphates*) and calcic and magnesic phosphates (*earthy phosphates*). It is derived from the food and from the retrograde metamorphosis of tissues containing phosphorus. The earthy phosphates are probably increased in diseases of the brain, osteomalacia, rachitis, etc. They are decreased in chronic spinal affections, dropsy, etc.

Test for the Earthy Phosphates. Fill a test tube ⅓ full with urine, and add a few drops of *caustic potash* or *ammonia* and heat until the phosphates precipitate in flakes. The test tube is then put on a stand for 15 minutes, and the quantity of the precipitate is determined. In an ordinary-sized test tube a deposit 1 cm. high represents a normal amount.

In health the phosphates are held in solution by an acid urine, and it is only when the urine has become neutral or alkaline that they are deposited.

Triple Phosphate. (Fig. 14 e.) When urea undergoes decomposition into ammonium carbonate, this with the magnesium phosphate in the urine, forms *ammonio-magnesium phosphate* (NH_4 Mg PO_4). Triple phosphate is recognized (Fig. 14 e.) by its large transparent crystals, occurring mostly in triangular prisms or feathery crystals. The crystals may be confounded with common salt and calcium oxalate. Common salt is only found in urine which has been concentrated by evaporation. Acetic acid will dissolve triple-phosphate, but not calcium oxalate. The crystals of triple-phosphate are usually present in cystitis associated with ammoniacal urine.

Neutral Phosphate of Calcium (Fig. 14 d.) forms wedge-shaped crystals, which unite to form rosettes.

Magnesium Phosphate forms long quadrilateral plates, with rounded ends.

Sulphuric acid (H_2 SO_4).—Daily amount excreted is about 2 grams (½ drachm). It appears partly as the sulphates of the alkalies, and a small portion as organic sulpho-acids. The sulphates are increased by an animal diet and exertion.

Test for the sulphates.—Acidulate the urine with a few drops of nitric acid and add a solution of barium chloride. A precipitate of barium sulphate is formed insoluble in water or acids.

Carbonic acid (CO_2).—Present in small quantities in human urine. Increased after vegetable food, certain drugs and in decomposed urine. Large quantities of the carbonates cause effervescence of the urine on the addition of acids.

Calcium carbonate (Fig. 14, f,) exceptionally, forms dumb-bell crystals. It is recognized by its effervescence and solubility upon the addition of mineral acids, which may be observed under the microscope.

ABNORMAL CONSTITUENTS OF THE URINE.

Albumen.—Albumen occurring in the urine (*albuminuria*), is usually pathological. The albumens found in the urine are ordinarily *serumalbumen* and *serumglobulin*. The quantity of albumen may vary from mere traces to 1–2% (rarely more.) The blood-serum contains about 5% of albumen. Albuminuria may be caused by affections of the kidney (*renal albuminuria*), *e. g.* acute and chronic nephritis, amyloid kidney; by hydræmic conditions of the blood (anæmia, leucæmia); in fever and acute poisoning; after epileptic attacks and apoplexy (*transitory albuminuria*).

Physiological albuminuria may occur in the female after suppression of the milk, after a diet rich in albuminous food (particularly a diet of eggs), increased renal blood pressure (after a cold bath), excessive mental or muscular exertion and in total absence of sodium chloride from the food.

Albumen may be present in inflammatory affections of the urinary tract below the kidney. Under these circumstances it forms part of such fluids as blood, pus and secretions from the generative organs; the quantity of albumen is usually small, and the formed elements (blood corpuscles, pus cells, etc.), usually indicate the source of the albumen .

Renal albuminuria is usually associated with a large quantity of albumen and the presence of casts. In

women albuminuria on account of contamination of the urine by vaginal discharge is comparatively frequent and errors may be averted by the use of a catheter.

Qualitative tests.—1. *Heller's test.* Add to the urine in a test tube some *nitric acid* allowing it to flow down the sides of the inclined test tube, so that the two fluids form separate layers; at their line of contact if albumen is present, there is formed a sharply defined ring-shaped cloudiness. In very concentrated urine, a precipitate may be caused by urates, but in this case, the ring is not so distinct and is more toward the surface of the urine. If warmed, the cloudiness produced by urates will disappear, but not so if due to albumen.

2. *Heat test.*—Heat the urine to the boiling point in a test tube and add one or two drops of *acetic acid* or 10–20 drops of *nitric acid.* If albumen be present a white opacity will appear. The addition of acid prevents the precipitation of the phosphates of the alkaline earths and favors the coagulation of albumen, which if present in small quantities, would not separate in an alkaline fluid.

If after the heat test the urine is allowed to stand for 24 hours, the quantity of albumen may be approximately determined from the degree of precipitation. A slight cloudiness but no precipitate occurs, when there is less than 0.01% of albumen present; 0.05% when the curved part of the test tube is barely filled with albumen; 0.1% when the coagulum in the test tube reaches $\frac{1}{10}$ the way up; 0.25%, ¼ the way up; 0.5%, ⅓ the way up; 1%, ½ the way up and 2%–3% when the whole fluid is completely coagulated.

3. *Acetic Acid and Ferrocyanide of Potassium.* Add to the urine 3–5 drops each of *acetic acid* and a 10% solution of *ferrocyanide of potassium*; the minutest traces of albumen are demonstrated by the formation of a white cloud. This test indicates the presence of albumen, globulin or albumose, but not peptone.

4. *Picric Acid.* The addition to the urine of a saturated solution of picric acid will produce a greenish cloudiness.

5. *Biuret Test.* The urine is rendered alkaline with *caustic potash* and 1—3 drops of a diluted solution of *sulphate of copper* are added. In the presence of albumen, the blue solution acquires a violet tinge.

6. *Salt and Hydrochloric Acid.* A saturated solution of common salt containing 2% of pure hydrochloric acid added to cold urine will produce a cloudiness.

7. *Metaphosphoric Acid.* One or two drops of this acid added to the urine will produce a cloudiness, even when mere traces of albumen are present.

8. *Potassio-Mercuric Iodide (Tanret's reagent).* Solution: Potassii iodid., 3.32 grams. Hydrarg. bichlorid., 1.35 grams. Aquæ destillat., q.s. ad. 100 cub. cent. First acidulate the urine with *acetic acid,* then add the double iodide solution drop by drop.

This will give a precipitate when there is only 1 grain of albumen to a quart of water.

Quantitative Tests.—(*Esbach's Albuminometer*). This consists of a test tube marked at its upper end with U and R and at the bottom with 1, 2, 3, 4, 5, 6, 7. The urine is added up to U and the reagent (picric acid 10 grams, citric acid 20 grams, water 1000 cub. cent.) to R. The tube is well shaken, and after standing 24 hours the level of the precipitate is read off, the number on the scale representing grams of albumen per liter. To obtain the percentage the respective figure is divided by 10. When the albumen is abundant the urine is first diluted with one or two volumes of water, and then multiplying the resulting figures by 2 or 3, as the case may be.

Tanret's method. To 10 cub. cent. of filtered urine add 2 cub. cent. of acetic acid diluting with a little water; next add Tanret's reagent (*see above*) drop by drop, counting the number of drops used; when the precipitate thus formed grows less, a drop of the urine is taken out and brought in contact with a few drops of a 1% solution of corrosive sublimate on a porcelain plate; if on mixing the two, a red precipitate occurs, the reaction is complete, and for each drop of the reagent used, less 3 drops allowed for excess, 0.5 grams of dry albumen per liter are present.

Method by Weighing. Pour 100 cub. cent. of filtered urine into a beaker and add a drop or two of acetic acid; heat on water bath for half an hour or more; collect precipitate, wash with alcohol and ether, then dry and weigh. The weight, less that of the filter, represents the percentage of albumen.

Peptones. Present in the urine (*peptonuria*) during the absorption of pus and the formation of exudations (pneumonia, abscesses, etc.).

Peptones are detected by the Biuret test after albumen or hemialbumose has been removed or proved absent.

Hemialbumose (*propeptone*). Rarely present in the urine, but found in osteomalacia and intestinal tuberculosis.

Test for Hemialbumose. Acidify the urine with a few drops of acetic acid and add $\frac{1}{8}$ its volume of a concentrated salt solution; boil and filter off the precipitate. Albumen and globulin remain on the filter. The filtrate is allowed to cool, and if a turbidity arises by the further addition of the salt solution, which disappears by heating and reappears on cooling, then hemialbumose is inferred to be present.

Fibrin is present in the urine in cases of hæmaturia, chyluria, tuberculosis of the genito-urinary tract, etc.

It is recognized by spontaneous coagulation in the urine.

Mucin.—Occurs in traces in normal urine. Increased in diseases of the urinary passages. It is precipitated by alcohol and dilute mineral acids but not by heat.

Test.—If acetic acid is added to cold urine a cloudiness or precipitate appears which is not dissolved by an excess of the acid if mucus is present.

Blood.—Blood in the urine, differs in appearance according to the part of the urinary tract from whence it is derived. If from the *renal parenchyma*, the blood is well mixed with the urine giving it a smoky appearance; it appears in small quantities and renal casts are present. From the *ureters*, long semicircular clots and strings are present. From the *urethra and bladder*, the quantity of blood is large and blood clots are present.

Tests for Blood. *Spectroscope.*—Two dark absorption bands in D and E of the spectrum, *i. e.*, in the yellow and green, the former being narrower, the latter broader (oxyhæmoglobin).

Heller's Test.—If the urine be heated with *caustic potash*, the earthy phosphates in precipitating carry down the blood-coloring matter and appear reddish brown instead of white.

Almen's Test.—Add to the urine a few drops of fresh *tincture of guaiac.;* after shaking, add a few drops of *resinous turpentine oil.* If hæmoglobin is present, the color will change to a distinct blue. The following mixture may be employed in place of turpentine oil: Glacial acetic acid, 30 drops; Distilled water, 15 drops; Oil of turpentine, absolute alcohol, chloroform,—of each 3 ounces (Hühnerfeld's mixture).

Microscopic Examination.—Teichmann's test (*See Blood*) may be conducted with the urinary sediment.

The most trustworthy evidence of blood in the urine even when the foregoing tests prove negative, is the recognition in the sediment of red-blood corpuscles under the microscope. When the corpuscles are pale as occurs in dilute urine, the addition of eosin or iodine in solution will deepen their color.

Bile.—Biliary matters in the urine may be caused by hepatogenic or hæmatogenic icterus (See pages 5 and 6). In the former, resulting from obstruction of the bile ducts, the *biliary coloring matter* as well as the *biliary acids* are present in the urine. In hæmatogenic icterus, the biliary coloring matter is present but not the biliary acids.

Tests for Biliary Coloring Matter.—Gmelin's test.—To the urine, *yellow nitric acid* is added in a manner to cause the two to form different layers; there will be a play of colors from green, blue, violet, red to yellow. The colors appear in the order mentioned. A blue ring alone may be caused by indigo, a reddish-brown one by hydrobilirubin and other substances.

Marechal's Test.—If a solution of iodine in iodide of potash be added to the urine, so that the two fluids may touch but not

mix, a green color will immediately develop below the layer of iodine.

If urine containing biliary coloring matter is shaken with *chloroform*, the latter becomes yellowish. The chloroformic solution of the bile pigment, may then be tested after Gmelin's method.

In all these tests, darkly-colored urine should be diluted with water before applying the test. Albumen does not interfere with the tests.

Tests for Biliary Acids.—Pettenkofer's test.—Unless applied in the following manner the results are usually negative: Evaporate about 6 ounces of the urine to dryness in the water bath. Extract the residue with absolute alcohol, filter, and add ether in excess (20 times the bulk of alcohol used). By this means the biliary acids are precipitated. The precipitate is removed by filtering, redissolved in distilled water and then decolorized by filtering through animal charcoal. The filtrate is now ready for the test; a single drop of a solution of syrup of cane-sugar diluted with water, is added to the filtrate in a test tube; sulphuric acid is then added drop by drop, while the test tube is immersed in cold water; a fine cherry-red or purple-violet color appears.

Method of Strassburg. Some cane sugar is dissolved in the urine, and a piece of filtering paper is then dipped into this and allowed to dry. If the filtering paper is now touched with concentrated *sulphuric acid*, a violet-red spot appears.

Pus.—Pus in the urine (*pyuria*) may arise from an inflammatory affection in some part of the urinary tract, or the communication therewith of abcesses. Pus arising in the *urethra* may be squeezed out by pressure, and usually escapes with the first portion of the urine. Pus from the *bladder* is accompanied with hypogastric pain, frequent urination, alkaline urine; and the quantity of pus is large. Pus from the *pelvis of the kidney* is small in quantity, and the reaction of the urine is usually acid. Pus from the *kidney* is small in quantity, and the urine is acid, as a rule; renal casts are found in the sediment, and the amount of albumen is always greater than can be accounted for by the pus present. There are also symptoms of renal disease. The localization of suppurative changes is aided by the character of the epithelium found in the sediment. In pyuria arising from affections above the bladder, there is a notable absence of mucus.

Tests.—Donné's Test. Pour off the supernatant urine, and to the sediment add liquor potassæ; the pus is changed to a gelatinous, ropy mass.

Microscope. Pus cells are ⅓ larger than red-blood corpuscles, and granular. Granules disappear, and a group of nucleoli appear, on the addition of acetic acid.

Grape Sugar.—(*dextrose*) $C_6 H_{12} O_6$, occurs in traces in normal urine (about 0.5 gram in 24 hours). The temporary presence of small quantities of sugar in the urine (under ½%) is without diagnostic importance, and occurs in health after a diet rich in carbohydrates (*transitory glycosuria*). It is persistently present in larger quantities in *diabetes mellitus.* The urine in this affection is increased in quantity, is clear, pale, of high specific gravity, and contains an increased quantity of urea. *Glycosuria* (temporary presence of sugar in urine) occurs: after poisoning (curare, nitrite of amyl, chloral, etc.), in acute infectious diseases, affections of the medulla oblongata, after epileptic attacks, etc.

Qualitative Tests. In the following tests if albumen is present it is first separated by boiling and filtration.

1. *Moore's Test.* Heat the urine a few minutes with ⅓ its volume of *liquor potassæ*, when, if sugar be present, a yellowish-brown color appears, which is darker the larger the quantity of sugar present. On adding *dilute sulphuric acid*, the smell of burnt sugar (*caramel*) is perceptible. This test is now little used, because, for its success, 0.3% of sugar must be present.

2. *Trommer's Test.* Add to the urine ½ or ⅓ volume of *liquor potassæ* and a few drops of a 10% solution of *cupric sulphate.* Heat test tube in its upper half to boiling, and if sugar is present, there will be a reddish-yellow precipitate of cuprous oxide.

3. *Fehling's Test.*—Solution; crystalline sulphate of copper, 520 grains, neutral tartrate of potash, 5½ ounces, officinal caustic soda solution, 3 ounces, distilled water to make 30 ounces.

This solution is diluted with 3 to 4 volumes of water, heated to the boiling point (should remain clear, otherwise to be discarded) and a little urine added. If sugar is present, a yellowish red precipitate of cuprous oxide will form.

Fehling's Method modified by Seegen.—Urine is filtered through a thick layer of animal charcoal (of blood) which absorbs the whole of the sugar; the charcoal is washed out with distilled water which dissolves out the sugar, which may be tested with Fehling's solution; traces of sugar may be found even in the 4th washing.

4. *Bismuth Test* (*Böttger*).—Add an equal quantity of *liquor potassæ* to urine and a small quantity of *bismuth subnitrate*; boil for a minute or two, and if sugar is present, the bismuth salt is reduced and a brown or black color is formed. With this test, albumen or sulphides in the urine, produce similar effects as sugar. *Nylander's modification* of this test consists in the use of the following solution: Bismuth subnitrate, 2 grams, rochelle salt, 4

grams, 8% solution of sodium hydrate, 100 grams. With this solution, urine containing sugar turns brown or black after boiling.

5. *Mulder's Test.*—After the urine is rendered alkaline with carbonate of sodium, a solution of *indigo carmine* (sulphate of indigo) is added until the urine turns blue. On heating, indigo blue is changed to indigo white and on exposure to the air turns blue again.

6. *Phenylhydrazin Test.*—This test is based on the power of *phenylhydrazine* to unite with grape sugar and form characteristic crystals. To a measuring beaker half full of water add 2 drachms of *hydrochloric phenylhydrazine* and three of *sodium acetate;* the compound having been heated, the same quantity of urine is added and placed in a vessel of boiling water for 15 minutes; it is then quickly put in cold water. After standing for some minutes, a yellow crystalline sediment of *phenylglucosazone* slowly falls. If only a little grape sugar is present, then the sediment under the microscope shows yellow rod like crystals terminating at each end in round balls or bunches.

7. *Molisch's Method —Menthol, thymol* or *alpha-naphthol* are used in their alcoholic solutions (1 to 7 alcohol) by mixing a few drops with the urine. To the mixture *sulphuric acid* is added so that the solutions do not mix; if sugar is present a red color (if thymol or menthol was used) is produced at the line of contact, or if alpha-naphthol was employed, a violet color with greenish borders.

8. *Johnson's Method.*—If a few drops of picric acid are added to the urine, which is then mixed with an alkaline hydrate, a deep red color appears if sugar is present. A light red color appears in normal urine.

The most reliable tests at present known to us, are Nylander's modification of the Bismuth test and the Phenylhydrazin test. In the former test, one part of the solution is taken to ten parts of the urine, which is boiled one minute, but never exceeding two minutes, when a dark solution will be obtained on cooling from the oxidation of the bismuth. This very sensitive test detects the presence of 0.08% of sugar.

Quantitative Tests for Sugar.—1. *Fermentation.* This test depends on the decomposition in the urine of sugar by the *torula cerevisiæ* (yeast plant) with corresponding reduction of the specific gravity of the fluid. After taking the specific gravity pour 4 ounces of urine into a bottle to which is added a piece of *yeast* about the size of a pea; the bottle is then loosely corked and set aside in a warm place for 24 hours, when fermentation will be completed. Allow the bottle to cool, and then take the specific gravity again. The difference gives the number of grains of sugar to the ounce.

Example:

Specific gravity before fermentation	= 1040
Specific gravity after fermentation	= 1015
Difference	= 25

25 grains of sugar are present to the ounce. The percentage is obtained by multiplying this number by 0.23. This method is the most certain test for sugar. It is well, in making the test to have two other bottles, one containing a solution of grape sugar and a little yeast (*to show that the yeast is active*), the other containing normal urine and yeast (*to show that the yeast is free from sugar*).

Fehling's method. Mix in a flask or beaker 10 c. c. of Fehling's solution (*see Fehling's test*) diluted with 40 c. c. of distilled water. Heat to boiling, and add from a burette containing a mixture of one part of urine and nine of water, a little at a time, stirring the mixture until the blue color of the test solution has entirely disappeared. As 10 c. c. of Fehling's solution are reduced by 0.05 grams of sugar, that amount of diluted urine which has reduced 10c. c. of Fehling's solution contained 0.05 grams of sugar. If 16 c. c. of diluted urine (1 in 10) were used, 1.6 c. c. of urine contained 0.05 grams of sugar. The percentage is obtained by the following proportion: $1.6 : 0.05 = 100 : \times$; $\times = 3$. 1 per cent.

By means of the *polariscope*, the quantity of glucose is readily ascertained, but the apparatus requires great care in adjustment, and errors are liable owing to the presence of other bodies allied to sugar.

Inosite ($C_6 H_{12} O_6$). Muscle sugar is present in the urine of renal disease and diabetes insipidus. It does not ferment, precipitate copper salts or affect polarized light.

Acetone ($CH_3 COCH_3$). May be found in normal urine. Increased in febrile diseases, in certain forms of diabetes, affections of the brain, etc. It is looked for in *diabetic coma*, in which condition it imparts a vinous odor to the breath and urine.

Tests. Perchloride of iron with acetone gives a Burgundy-red color (unreliable).

Lieben's Test. Twenty grains of *potassium iodide* are dissolved in one drachm of *liquor potassæ* and boiled. The urine is poured on the surface of this solution producing at first a precipitation of the phosphates, but if acetone is present the deposit becomes yellow, owing to the development of *iodoform*, which is recognized by its characteristic odor.

Legal's Test. Add to the urine a few drops of freshly prepared *nitro-ferrocyanide of sodium* and then *caustic soda* until it is strongly alkaline. When the beginning purple tint turns yellow, 1–3 drops of *acetic acid* are added, and if the acetones are present a crimson-purple color is formed at the point of contact of the acetic acid and the mixture.

Ehrlich's Diazoreaction.—This reaction present in typhoid fever and in relapses of this affection is of some value in doubtful cases. Its disappearance is a good sign.

It is also present in severe cases of phthisis, pneumonia and measles.

Test. Solution 1. Sulphanile acid, 5 grams, muriatic acid, 50 grams, distilled water, 1000 grams. *Solution* 2. Nitrite of sodium 0.5 grams, water, 100 grams. Take 50 ccm. of solution 1 and 1 ccm. of solution 2 and mix in a test tube with an equal quantity of urine to which is added ⅛ volume of ammonia, and the whole well shaken. The reaction is positive when a red color is formed, especially noticeable in the foam.

The *"Diazo" reaction* constitutes one of the earliest and most constant signs of typhoid fever. Febrile abdominal catarrh never shows this reaction. Disappearance of the reaction points to approaching apyrexia. Relapses are almost invariably accompanied by its reappearance.

Sulphuretted Hydrogen (H_2S). Present in decomposed urine.

Test. Filtering paper saturated with acetate of lead solution when held over the bottle turns black (*formation of lead sulphide*).

Cystin. Occasionally found in urine. Under the microscope it appears as colorless, shining, six-sided plates or prisms (Fig. 14).

Leucin and Tyrosin. Found in acute yellow atrophy of the liver and in phosphorus poisoning.

Under the microscope *leucin* appears as yellow-colored spheres, at times concentrically striated with protruding points (Fig. 14).

Tyrosin appears in white needles arranged in bundles. To obtain these crystals for microscopical examination the urine is evaporated to a syrupy consistency.

ORGANIC SEDIMENTS.

White Blood Corpuscles (leucocytes). Normally present in small numbers. Increased in inflammation and suppuration of the genito-urinary tract (nephritis, pyelitis, cystitis, urethritis and leucorrhœa).

Red Blood Corpuscles. Present in hæmaturia. They are usually pale, and may appear as casts.

Renal epithelium. Round or cuboid cells with a vesicular nucleus and often full of fat drops (Fig. 15, i).

Arranged in cylindrical form they make epithelial casts. Renal epithelium nearly always indicates an affection of the kidney.

Epithelial cells from the *renal pelvis*, *ureters* and *bladder* cannot be differentiated from each other. The cells of the superficial layers have a polygonal form, while those of the deeper layer are round, with processes, and contain a vesicular nucleus. An increase of these cells in the urine may indicate cystitis, pyelitis or inflammation of the ureters. The *vagina* and *prepuce* have pavement, and the *male urethra* cylindrical epithelium.

Casts. These are moulds of the renal tubules produced by the escape of a coagulable material which coagulates and entangles other substances, and are characteristic of renal disease. They may be divided according to composition and appearance into three groups (v. *Jaksch*). 1. Cellular. 2. Those composed of products of degeneration. 3. Hyaline.

1. The cells found in casts are red-blood corpuscles, leucocytes, renal epithelium and sometimes colonies of bacteria.

2. The products of degeneration are granules derived from epithelial *débris*, amyloid material and fat.

3. Hyaline cylinders are composed of the ground substance only.

Blood Casts (Fig. 15, b) are red-blood corpuscles held together by coagulation. They are a certain sign of *renal hæmaturia*.

Leucocyte casts may occur in purulent affections involving the renal tubules.

Epithelial casts (Fig. 15, a) are composed essentially of renal cells embedded in a hyaline albuminoid substance.

Casts of Bacteria are present in septic embolic nephritis or pyelo-nephritis. With a low power they resemble granular casts.

Granular casts (Fig. 15, d) are composed of granules, which consist of broken-down blood or epithelial cells.

Fatty casts are usually hyaline casts dotted with oil globules. Any cast covered with oil drops is known as a "fatty cast." These casts may indicate fatty degeneration in the kidney.

Amyloid casts are transparent, homogeneous and very brittle, and stain deeply with a solution of iodine.

Hyaline casts (Fig. 15. c) are colorless, very long and narrow. They are easily overlooked, and it is advisable in searching for them as well as other casts to add a drop of Lugol's solution or

aniline red to make them more distinct. These casts have been occasionally found in normal urine.

Mucous casts of the uriniferous tubules are usually very long, and when present do not necessarily indicate disease.

Casts of the seminal tubes are very rare, and are usually of greater calibre than renal casts.

Casts of Ammonium Urate (Fig. 15, f) are commonly found during the first days of life in children. Treated with potassium hydrate, ammonia escapes, and the casts disappear.

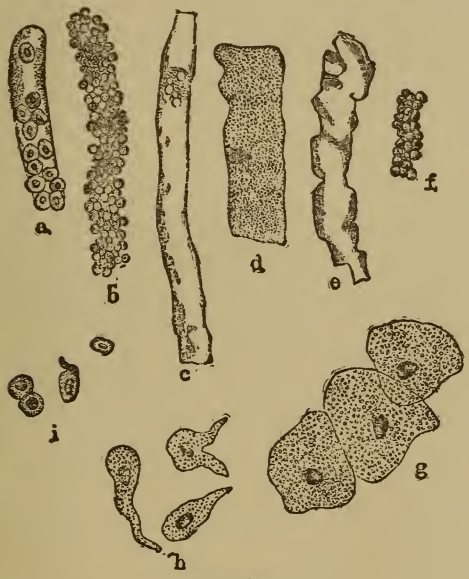

Fig. 15.

Fig. 15. a. Epithelial cast. b. Blood cast. c. Hyaline cast. d. Granular cast. e. Waxy cast. f. Cast of ammonium urate. g. Pavement epithelium from the bladder (upper layer). h. Same (lower layer). i. Epithelial cells from the uriniferous tubules.

Spermatozoa may be found after coitus, nocturnal emissions, spermatorrhœa, etc. Found in the urine of women they may be of medico-legal importance. *Fragments of morbid growths* may be met with in the urine.

Micro-organisms are present in many infectious diseases (diphtheria, recurrent fever, etc.).

Tubercle bacilli are present in tuberculosis of the genito-urinary tract, and *gonococci* in gonorrhœa.

In examining for the tubercle-bacilli, the method is the same as that detailed elsewhere (*see Sputum*). A number of cover-glass preparations from the sediment of the urine must be made.

Animal parasites are occasionally found in the sediment. The shreds or hooklets of echinococci occur in the urine when this parasite affects the kidney.

The *Distoma hæmatobium* inhabits the small veins of the kidney and portal system and the ova are expelled with the urine accompanied by blood.

In *chyluria*, the *Filaria sanguinis* has been found at times in the urine, but it is more constantly present in the blood.

Non-pathogenic organisms develop in urine exposed to the air. In urine containing sugar, the yeast plant (*Torula cerevisiæ*) often occurs.

SECRETIONS OF THE MALE GENERATIVE ORGANS.

Semen. Semen is a complex fluid composed of secretions of the testicles, of the seminal vesicles, of the accessory glands of the urethra, especially the prostate, Cowper's glands, and the glands of the mucous membrane of the urethra. With the microscope, spermatozoa, spermatic cells, epithelium from the prostate and urethra and seminal granules can be distinguished. In fresh semen, the motion of the spermatozoa is very active. The amount of semen discharged at a single evacuation varies from 5–10 grams.

Pathological Changes. Absence of semen (*Aspermia*) may be congenital or acquired. Acquired permanent aspermia may follow occlusion of the ejaculatory ducts or destruction of the glandular tissue of the prostate gland. Temporary aspermia may occur with a comparatively normal state of the sexual apparatus.

Polyspermia. An increase in the amount of semen discharged at a single ejaculation is relatively seldom observed.

Oligospermia signifies a decrease in the amount of semen discharged. Observed in advanced age, and after inflammation of the testicles and diseases of the prostate.

Oligozoöspermia. Signifies a diminution in the number of spermatozoa, while azoöspermia means their total absence. Oligozoöspermia is most frequently caused by gonorrhœal inflammations of the epididymes and spermatic cords.

	easily; *sweet* taste. As a rule no *sediment*. Yeast *fungus* appears in urine after exposure to *pericystitis* to the urine.	*ritis*, urine is light colored; in *chronic Parenchymatous Nephritis*, dark; in *acute Nephritis*,	light. Urine looks like porter. Stains linen yellow. Yellowish foam (*Santonin* and *picric*	*Fuchsin*, and other like vegetable coloring matter may produce a reddish urine, but color		metal successively with *hot water*, *alcohol*, & *ether*. Now put the metal into a acid.
Microscopical Examination.	The yeast fungus (*torula cerevisiæ*) and other forms of fungi, found even in fresh urine	*Renal Albuminuria*; *Kidney casts* and *Epithelial cells* (small, round, or cuboid with a yes- albumen to disap-	*Hyaline cylinders* and blood-stained epithelial cells. The cylinders are probably the result	*Renal Hæmaturia.* Blood casts and renal Epithelium. The kind of Epithelium in the sed-	*Uric acid*, *Ammonium Urate*, lozenge-shaped globules with projections. *Calcium Oxalate*, dumb-bell and	*Santonin.* Urine (straw yellow) turns *red* on adding alkalies.

poses.

Medicinal: Opium, codeia, bromides. Antipyrin is said to possess anti-glycogenic properties (daily dose, 45 grains). Iodoform (gr. ½ to 2 grains 3 times a day) diminishes excretion of sugar. Ergot, arsenic and jambul seeds are also used. *Iodides* in diabetes of syphilitic origin. Tonics, alkaline mineral waters.

pear from the urine. Tannic acid, ergotine, fuchsin, acetate of lead and other astringents are sometimes useful. The *iodides* in *interstitial nephritis.*

of gall bladder, use the faradic current and massage in this region. Mineral acids.

Mineral acids.

In *alkaline lithiasis* render urine acid.

Rhubarb and

Pus is detected by adding an equal amount of Liquor Potassæ to sediment of urine, and the same will become gelatinous, indicating pus. *Mucus* in urine is precipitated upon the addition of Acetic Acid. About 50 grams of solid matter is excreted in 24 hours. One-half of this consists of Urea, and one-fourth of Sodium Chloride. In order to estimate approximately the quantity of solids excreted in 24 hours in 1000 parts of urine, multiply last two figures of specific gravity by 2 (e. g., 1500 cbc. of urine with a spec. gr. of 1017= 2×17=34 grams in 1000 cbc. In 1500 cbc.=34+17=51 grams.) The excretion of the Chlorides is diminished in febrile diseases, especially in Pneumonia. Normal amount reappears in resolution. It may be roughly detected by adding a few drops of Nitric Acid to urine and then a few drops of a 10 per cent. solution of Nitrate of Silver; precipitate is soluble in Ammonia. Normal urine treated in a similar way may be used as a basis of comparison.

Indican results from the decomposition of albumen in the intestinal canal, and from its amount in the urine a conclusion may be drawn as to the intensity of the decomposition. It is increased by an abundant meat diet, abnormal putrefaction of the ingesta, and is most increased in intestinal obstruction. The following reaction is only positive when indican in the urine is increased (*Indicanuria*). Add to urine an equal quantity of hydrochloric acid and 1 or 2 drops of a concentrated solution of *calcium chloride* which is one-half diluted with water. The chloride of calcium solution is added drop by drop until a blue color appears. A few ccm. of chloroform are then added, and the whole shaken, which brings out the indigo.

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*A synopsis of morbid renal secretion (Revised second Edition): 1890.

A SYNOPSIS OF MORBID URINE.*

Normal Urine is of acid reaction. Specific gravity, 1015-1020, at a temperature of 60° F. Transparent amber-yellow colored fluid of consistency of water; mild taste; peculiar odor, usually excreted in 24 hours of from 40-50 ounces by men (more when increased by women (1000-1500 ccm. (30-40 ounces). Under 500 ccm. (15 ounces) and over 3000 ccm. (90 ounces) is usually pathological.

	SUGAR.	ALBUMEN.	BILE.	BLOOD.	UNDESTROYED SEDIMENTS.	DETECTION OF SUGAR.
	1020-1070. In rare cases of *Diabetes insipidus*, the specific gravity may be low, even 1005-1010.	Sp.gr. in *Interstitial Nephritis* rarely above 1012. In acute *Nephritis*, above 1020. In chronic *Nephritis*, above 1025.	Normal or slightly increased.	Increased or diminished. In malarial poisoning, in cases of cancer, in the earlier periods in the various combination with the blood corpuscles.		*Iodide of Potassium.* Mix urine with a few drops of fuming *Nitric acid*. Shake. The volume of the urine should be about the Chloroform will show a reddish-violet tint.
Specific Gravity.	1040-1060. Examination of urine passed in 24 hours, and take the sp. gr. Individuals vary. Measure urine passed in 24 hours, and take the sp. gr. Multiply the number of ounces by the number of degrees of specific gravity left off. Gives the percentage amount of sugar present.	*Hæmoglobinuria*—When blood-containing matter sides out of corpuscles is present.				*Bromide of Potassium.* Add to urine *fresh* chlorine water and *Chloroform*, and shake. The Chloroform will show a yellow color.
Reaction with Litmus.	*Acid.* Relative solidity a loss than 1 gm. to 1 oz. nol red formation takin place.	*Acid* as a rule in spite of the *Nephritis*, acid, the urine is alkaline.	Usually acid.	As a rule, acid-alkaline, when treated in reactive to acetic acid, but *Chloride of Ammonium* is not changed. To determine whether alkaline reaction is due to alkaline *Carbonates* of sodium (*Potassium*) or *fixed alkalies*; the former causes the blue color to disappear, the latter remaining when due to fixed alkalies.			*Iron.* When present in large quantities the solution of iron. *Sulphite* throws a greenish-black color.
				In acid urine.	*In alkaline urine.*	*Acetic Acid* causes a white precipitate with *Nitric acid* and *Chloroform*, and shaken, the Chloroform will show a yellow color.
				AMORPHOUS.		
				URATE.	CALCIUM URATE. PHOSPHATE.	
				SODIUM URATE.	CRYSTALLIZED.	
				CALCIUM OXALATE.	AMMONIUM URATE.	
				CYSTIN.	MAGNESIUM PHOSPHATE.	
				LEUCIN.	CALCIUM CARBONATE.	
					MAGNESIUM PHOSPHATE.	
					CALCIUM PHOSPHATE.	
					AMMONIUM URATE.	
					TRIPLE PHOSPHATE.	
					CALCIUM CARBONATE.	
					URATE.	
					CALCIUM OXALATE.	
					CYSTIN.	
					LEUCIN.	
					TYROSIN.	
					CALCIUM PHOSPHATE.	
					AMMONIUM URATE.	
					CALCIUM CARBONATE.	
					MAGNESIUM PHOSPHATE.	
					URIC ACID.	
					AMMONIUM URATE.	
					CALCIUM OXALATE.	
					CYSTIN.	
					LEUCIN.	
					TYROSIN.	
					CALCIUM PHOSPHATE.	
					CALCIUM CARBONATE.	
					MAGNESIUM PHOSPHATE.	
					TRIPLE PHOSPHATE.	
					CALCIUM OXALATE.	
					CYSTIN.	
					LEUCIN.	
					TYROSIN.	
					CALCIUM PHOSPHATE.	
					CALCIUM CARBONATE.	
					MAGNESIUM PHOSPHATE.	
					URIC ACID.	
					AMMONIUM UR

Secretion of the Prostate. This is a milky fluid showing under the microscope cylindrical epithelial cells white blood corpuscles, amyloid bodies and glistening granules. If a few drops of a 1% solution of phosphate of ammonia is added to the fluid under the microscope, *spermatic crystals* are formed.

Prostatorrhœa signifies involuntary loss of the prostatic secretion. It occurs in all irritable conditions of the prostate, whether produced by gonorrhœa, masturbation or senile hyperplastic alterations of the gland.

Secretion from the Glands of Cowper is an odorless, clear viscid fluid, showing under the microscope epithelial and round cells. This secretion normally appears during erection of the penis as a clear, transparent drop at the meatus.

CHAPTER XII.

THE NERVOUS SYSTEM.

Anatomy and Physiology of the Brain and Spinal Cord.—The cerebral cortex *i. e.* the gray matter upon the surface of brain is the seat of mental activity. It has ridges (*gyri or convolutions*) and depressions (*sulci or fissures*). The *psycho-motor region* of the

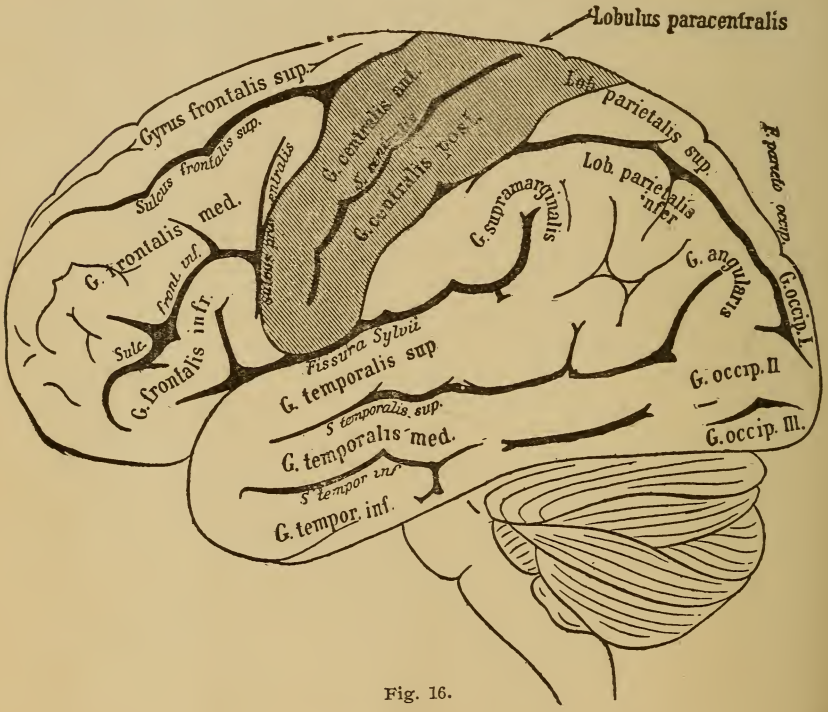

Fig. 16.

cortex (*motor area*) comprises the *anterior* and *posterior convolutions* and the *lobus paracentralis* of either hemisphere (*this area is shaded in Fig.* 16).

When certain limited areas of the brain cortex are stimulated by electricity in animals, according to the area stimulated, movements of definite muscle-groups can be evoked, which is not the case when these areas are extirpated.

The excitable areas of the cortex are as follows :

1. The *region of the arm* lies in the middle third of the anterior convolution (*G. centralis ant.*)
2. The *region of the leg* lies in the upper third of the central convolutions and the lobus paracentralis.
3. The *region of the head* lies in the lower third of the central convolutions and that portion of the frontal convolution bordering on the fissure of Sylvius.
4. *Region of the trunk*, convex surface of the frontal lobe bordering on the præcentral fissure.
5. *Center of vision*, occipital lobes.
6. *Center of hearing*, temporal lobes.
7. *Center of speech*, inferior frontal convolution (*left side*) and adjacent area, around the lower part of the fissure of Sylvius.

The cortex of the *parietal lobes* is associated with the sensory tract.

Pyramidal Tract (Fig. 17). From the cortical centres the motor fibres converge toward the large ganglia at the base of the brain, where they present a white strand of fibres (*internal capsule*).

That portion of the capsule in front of the head of the caudate nucleus and behind the lenticular nucleus, constitutes the *anterior limb* of the internal capsule. As the capsule passes along the internal margin of the lenticular nucleus it makes a bend (*genu*) and runs between the front of the optic thalamus and behind the lenticular nucleus, forming the *posterior limb* of the internal capsule. From the internal capsule, the motor fibres pass into the pons and from the pons into the medulla forming the *pyramidal bodies*.

At the lower part of the medulla, about 10% of the fibres pass down the same side of the spinal cord in the anterior pyramidal tracts, whereas the other fibres (*crossed fibres*) pass to the opposite side of the cord into the lateral pyramidal columns. From these columns the fibres pass into the ganglionic cells of the anterior horns, and from these cells the anterior roots of the spinal cord are formed which send motor fibres to the muscles. The centers of the cerebral cortex control the nutrition of the pyramidal tracts up to the point where they enter the ganglia of the anterior horns of the cord, whereas the ganglia of the anterior horns control the nutrition of the peripheral motor nerves and muscles.

Lesions of the motor tract result not only in motor paralysis but also in descending degeneration of the pyramidal tracts, inasmuch as their trophic center is in the cerebrum. The trophic center of the peripheral motor nerves is situated in the ganglionic cells of the anterior horns, hence lesions here result in degeneration of the nerves, and paralysis and atrophy of the affected muscles.

The Sensory or Centripetal tract takes its origin in the sensory nerves of the periphery which reach the cord through the posterior or sensory roots which on their entrance immediately cross over to the opposite side.

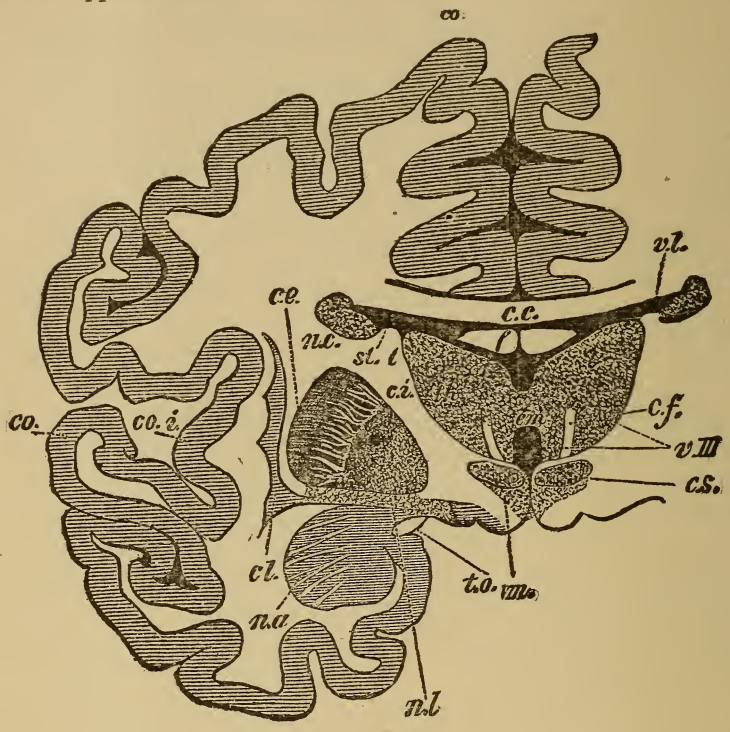

Fig. 17.

Fig. 17. Diagrammatic Transverse Vertical Section of the Cerebrum—(*After Schwalbe*).

co, cortex
coi, island of Reil
cl, claustrum
na, nucleus amygdalæ
nc, caudate nucleus
th, optic thalamus
cm, middle commisure
cs, subthalamic body
m, substantia nigra
nl, lenticular nucleus
ci, internal capsule
ce, external capsule
stt, stria terminalis (tænia semicircularis)
cf, anterior pillar of the fornix
f, fornix
cc, corpus callosum
vIII, third ventricle
vl, lateral ventricle
to, optic tract

The *blood supply of the brain* is derived from the *internal carotid* and *vertebral arteries*. Each carotid divides into the anterior cerebral and middle cerebral arteries. The *middle cerebral artery* enters the fissure of Sylvius and sends branches to the lenticular nucleus and adjacent parts of the brain, including the caudate body of the corpus striatum, optic thalamus and internal capsule. The anterior and middle cerebral arteries do not freely anastomose, and are called *terminal arteries*.

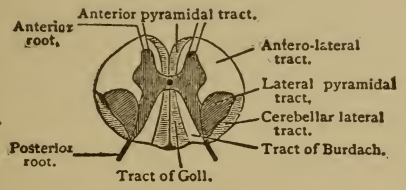

Fig. 18.

Spinal Cord (Fig. 18). The *Anterior* and *lateral pyramidal tracts* of each side contain only *motor fibres*. Those in the former (*Türk's columns*) are connected with the corresponding cerebral hemisphere; the latter (*crossed pyramidal tracts*) with the opposite cerebral hemisphere. The *posterior columns* (*tracts of Goll* and *Burdach*) convey sensory nerve fibres.

Diagnosis of Brain Lesions.—*Lesion of the internal capsule:* there is hemiplegia or hemianæsthesia (loss of sensation in one lateral half of body) or both combined; hemiparalysis of the lower half of the face. Tremor, hemichorea and athetosis.

Lesions of the crus cerebri: if the lesion is confined to the *tegmentum cruris* (sensory portion), there is hemianæsthesia of the opposite side of the body and paralysis of the 3d and 5th cranial nerves on the same side. If the motor portion (*crusta cruris*) is alone implicated, there is paralysis of the 3d nerve with hemiplegia on the opposed side.

Corpora Quadrigemina : incoördination of movement, absence of pupillary reflex, nystagmus and strabismus. Blindness may exist independent of a choked disc, optic atrophy or neuritis. *Pons varolii;* lesions may be above or below the *line of Gubler*. This is an imaginary line, that connects the apparent origin of the trigeminal roots and marks the level of the decussation of the facial nerve

fibres that pass cephalad. *Lesion above the line of Gubler:* facial paralysis and hemiplegia upon the same side of the body and opposed to the seat of lesion. *Below the line of Gubler:* facial paralysis on the same side as the lesion and hemiplegia on the opposed side. *Cerebellum:* incoördination of movement, intense vertigo and titubating gait. Only when the vermiform process is implicated are the symptoms of cerebellar lesions pronounced. *Medulla oblongata:* disturbances of respiration, circulation, phonation, deglutition and articulation (when cranial nerve roots are implicated). Diabetes and albuminuria may be present. *Lesions at the base of the brain (anterior fossa):* implication of the olfactorius. *Middle fossa:* implication of the opticus, oculomotorius, trochlearis and abducens. *Posterior fossa:* implication of the trochlearis, abducens, facialis, acusticus, glosso-pharyngeus, vagus, accessorius and hypoglossus. Basal lesions may also involve the pyramidal tracts.

Lesion of the thalamus opticus: post-hemiplegic chorea, athetosis, and tremor of the paralyzed limbs. Hemianopsia.

Diagnosis of Spinal Lesions.—Lesions of the cord before *pyramidal decussation* cause paralyses of the same side. Usually lesions of the spinal cord affect both sides alike leading to *paraplegia*, a frequent form of spinal paralysis. In cases of *transverse section* of the cord, the *columns of Goll* and *cerebellar lateral tracts* degenerate upward from the point of injury and the *pyramidal columns* downward. Lesions of the spinal cord are *systematic* and *non-systematic* or focal. The former refer to affections limited to recognized subdivisions of the spinal cord without spreading to adjacent parts; by the latter is meant affections not limited but which spread as the lesion progresses. In *Tabes dorsalis* the *lesion* is in the posterior columns; *Lateral sclerosis:* lateral columns, *Amyotrophic lateral sclerosis:* anterior cornua and lateral columns; *Acute anterior poliomyelitis* (infantile spinal paralysis): anterior cornua; *Progressive muscular atrophy:* anterior cornua. In *bulbar paralysis* there is degenerative atrophy of the nuclei of nerves of the medulla oblongata.

Paralysis.—When the movement of an extremity is entirely absent, the term *paralysis* is used; when slight movement is possible, *paresis.*

Paralysis may be *cerebral*, *spinal* or *peripheral.*

Hemiplegia signifies paralysis in one lateral half of the body. *Paraplegia* signifies paralysis of both legs or arms. *Hemi-paraplegia:* paralysis of motion in the lower one-half of one lateral half of the body. *Monoplegia:* paralysis of a distinct group of muscles. *Central paralysis:* lesions of the motor tracts proximal to the anterior horns of the cord. *Peripheral paralysis:* lesions of the motor nerves peripheral to the anterior horns of the cord. *Flaccid paralysis:* when the paralyzed limb readily yields to passive movement. *Spastic paralysis:* when the paralyzed limb offers a resistance to passive movement. Spastic paralyses are usually associated with exaggerated reflexes. *Functional paralysis* (no anatomical lesion): are usually unattended by trophic or electrical disturbances.

Cortical Paralysis.—This is due to some lesion (abscess, blood clot, tumor, embolism, meningeal thickening, depressed bone, etc.,) of the cerebral cortex. Cortical lesions may be *irritative* or *destructive.*

Irritative lesions are usually connected with paroxysmal cramps in some special group of muscles (*cortical or Jacksonian epilepsy*). Paralysis if present is usually transient and returns after subsequent paroxysms.

In destructive lesions there is paralysis of special groups of muscles (*monoplegia*). Consciousness is not lost as in lesions of central portions of the brain. *Pain* within the head in these lesions is usually localized or percussion over the seat of the lesion will elicit it. No impairment in sensibility of the paralyzed parts exists, unless sensory paralysis is present as a complication. Affected muscles show normal electro-contractility. Paralysis (as in other cerebral lesions) is on the side opposite to the lesion.

Hemiplegia (*seat of lesion*). If intra-cranial, the paralysis is on opposite side of body; if spinal, on the same side.

Symptoms.	*Seat of Lesion.*
Hemiplegia with motor aphasia.	3d left frontal convolution.
" " paralysis of lower facial branches.	Posterior limb of internal capsule.
" " hemianæsthesia.	Posterior third of internal capsule.
" " crossed paralysis of 3d cranial nerve.	Crus cerebri on same side as paralyzed nerve.
" " crossed facial paralysis.	Pons.

Etiology. 1. Embolism. 2. Apoplexy. 3. Syphilis. 4. Toxic. In *embolism*, the paralysis is usually sudden without loss of consciousness and usually associated with *motor aphasia*. There is usually a heart lesion. Patient usually young.

Apoplexy. Origin sudden with loss of consciousness and usually associated with lower facial paralysis. Arteries atheromatous and patient is usually old. In the *stage of reaction*, there is a rise of temperature (from inflammatory changes around the clot.)

Syphilis. Origin slow. Lesions multiple. History of syphilis. Good results from anti-syphilitic treatment. Occurs in the young.

Toxic. In uræmia and the later stages of phthisis and carcinoma (hemiplegia is transitory).

Paraplegia (*seat of lesion*). When the paralysis is incomplete the term *paraparesis* is used.

Lesion may affect lumbar (frequent), dorsal or cervical region (paralysis of all four extremities).

Etiology. Myelitis, trauma, carcinoma, tuberculosis, caries and syphilis.

PARALYSIS OF THE CRANIAL NERVES

1. *Olfactorius.* Loss of smell (*anosmia*). Test with asafœtida, etc.

2. *Opticus.* Diminished sharpness of vision and sense of color; narrowing of the field of vision (examine with ophthalmoscope).

Loss of vision in one lateral half of each retina is called *hemianopsia.* The most common form is *homonymous hemianopsia*, when nasal half of one eye and temporal half of the other eye is blind. This may result from some lesion of the optic tracts or the cortex of occipital lobe. The *binasal variety* indicates a lesion of the *chiasm. Total blindness* (amblyopia or amaurosis) is due to a lesion of the opticus peripheral to the chiasm.

Hemianopsia may be detected by requesting the patient to close one eye and to fix the open eye on some near object. Now take some object easily seen and move it to the right and left and also above and below the object upon which the patient is gazing, asking the patient in each case if the two objects are seen distinctly and simultaneously. The retina is blind upon the side opposite to that upon which the moving object is lost to sight.

3. *Oculomotorius.* Falling of the upper eyelid (*ptosis*), external strabismus, dilatation and absence of the pupillary reaction, double vision (*diplopia*) and disturbances of accommodation.

4. *Trochlearis.* Eye cannot be raised or rotated outwards.

5. *Trigeminus.* Anæsthesia of the sensory distribution of this nerve; paralysis of the masticatory muscles (motor fibres) and impairment of taste (lingualis to ant. ⅔ of tongue).

6. *Abducens.* Eyeball cannot be turned outward.

7. *Facialis.* Immobility of the muscles of expression of one side of the face; eye cannot be closed, the face is drawn to the opposite side, dribbling of saliva, etc. In lesions proximal to the *ganglion geniculi*, the soft palate on the same side is paralyzed; if lesion is between the *ganglion geniculi* and the passage of the *chorda tympani*, there are disturbances of taste in the anterior ⅔ of tongue. In *central paralysis of the facialis*, only the lower half of the face is usually paralyzed, the electro muscular contractility is unchanged and hemiplegia is frequent. The *prognosis of peripheral facial paralysis* is influenced by the electric reaction. If the electric reaction is normal, recovery can be expected in 2 or 3 weeks; if diminished but not lost, recovery in 6 weeks; loss of the faradic and galvanic excitability of the nerve and loss of the faradic excitability of the muscles (*reaction of degeneration*), gives an unfavorable prognosis.

8. *Acusticus.* Disturbances of hearing (examine with the otoscope).

9. *Glossopharyngeus.* Loss of taste in the posterior ⅓ of tongue.

10. *Vagus.* Quickening of the pulse and slowing of the respiration.

11. *Accessorius.* Paralysis of the sterno-cleido-mastoid and trapezius.

12. *Hypoglossus* (motor nerve of tongue). Tongue turns toward the paralyzed side when protruded.

PARALYSIS OF THE SPINAL NERVES.

Plexus Brachialis. Paralysis of the deltoid, biceps, brachialis anticus, supinator longus and infraspinatus (*paralysis of Erb*) muscles.

Nervus Medianus. Pronation and flexion of the hand, flexion and opposition of the thumb and flexion of the finger in the last two phalanges is impossible.

Nervus Ulnaris. Power is diminished to flex and draw the hand to the ulnar side as well as to flex the last 3 fingers. Little finger is immovable. On account of atrophy of the *interossei*. the first phalanges are extended and the second and third phalanges are flexed (*claw-hand*).

Nervus Radialis (musculo-spiral). Hand hangs in flexion and cannot be extended. Fingers are flexed. Thumb cannot be extended or abducted. Outstretched arm cannot be supinated,

but on flexion of the arm the forearm can be supinated by the biceps. *Lead poisoning* is a frequent cause of paralysis of this nerve, but the supinator longus is not usually involved.

Nervus Peroneus. Flexion of the foot is impossible. Tips of toes and the outer edge of foot first touch the ground in walking (abduction of these parts abolished).

Nervus Tibialis. Extension of the foot is lost (patient cannot rise on his toes). Abduction of the foot and plantar flexion of the toes (*claw-like appearance*).

ATAXIA. Inability to coördinate with intact muscular power certain muscular movements. If patient is directed to thread a needle, pick up a pin or make other complicated movements, the motions are clumsy. Ataxia occurs in cerebral and spinal affections (*tabes*), affections of cerebellum and in diseases of the peripheral nerves (*neuritis*) following diphtheria, alcoholism, etc.

APHASIA. *Ataxic* (motor) *aphasia*, the power of coördinating the movements for articulate speech is partially or totally lost.

Amnesic (sensory) *aphasia*; the power of recollecting words as aggregate acoustic phenomena is impaired.

Word dumbness; the inability with intact hearing to understand words. This is also called *word deafness.*

Paraphasia; the ability to connect word images and corresponding connection is lost.

Agraphia; this is the inability to convey thoughts in writing. It is present in amnesic aphasia.

The lesion in aphasia is most frequently found in the left hemisphere in the inferior frontal convolution (*Broca's convolution*).

TESTING THE SENSIBILITY.

Touch Sense. This signifies (*eyes closed*) the ability to immediately perceive the contact and describe the sensation, when the skin is touched with the finger or any other object.

Sense of Locality. Normal individuals locate almost accurately the part of the body touched. By the use of compasses (*æsthesiometer*), the smallest distance may be

found in which both points may be recognized. The distance varies in health, and the following measures can be used as the healthy standard for comparison: 1. Point of tongue, $\frac{1}{40}$ inch. 2. Tip of finger, $\frac{1}{20}$ inch. 3. Mucous surface of lips, $\frac{1}{13}$ inch. 4. Tip of nose, $\frac{1}{6}$ inch. 5. Chin, $\frac{1}{4}$ inch. 6. End of big toe, cheek and eyelids, $\frac{1}{3}$ inch. 7. Bridge of nose, $\frac{1}{3}$ inch. 8. Heel, $\frac{1}{2}$ inch. 9. Back of hand, $\frac{3}{4}$ inch. 10. Neck, $\frac{7}{8}$ inch. 11. Fore-arm, $1\frac{3}{5}$ inch. 12. Sternum, $1\frac{4}{5}$ inch. 13. Middle of thigh, $2\frac{1}{2}$ inches. 14. Back, $2\frac{3}{5}$ inches.

Sense of Pressure. This may be tested by placing different weights on the extremity. When the sense of pressure is greatly disturbed, it can be determined by pressure with the finger.

Sense of Temperature. May be tested with test tubes filled with water of different temperatures applied to the skin. It may be rapidly tested by breathing and blowing on the skin.

Electro-cutaneous sensibility. Tested with a wire-brush electrode and determining the minimum strength of the current felt.

Joint and Muscular sense. This is the ability to determine the position of the extremities and their passive movements with closed eyes.

Sensibility to Pain. Tested by sticking with a needle, electric current, etc.

Abnormal Conditions of Sensation.—*Anæsthesia* signifies entire loss of sensibility and depends on interruption of sensory conduction. *Hypæsthesia*, diminished sensibility. *Hyperæsthesia*, increase of sensibility. It is present in tetanus, hydrophobia, strychnia poisoning, hysteria, etc. *Hemianæsthesia*, loss of sensation in one lateral half of body. Present in lesions of the posterior third of the internal capsule and in hysteria. *Polyæsthesia*, is the perception of more than one impression although only one is made.

Delayed sensation, is an evidence of imperfect conduction of sensations to the brain and is a frequent symptom of tabes. *Analgesia*, is the absence of pain when the skin is

pricked with a needle. Occurs with unimpaired tactile sense in hysteria and tabes. *Perverse sense of temperature* is present when anything cold is perceived as warm. *Symptom of Romberg;* the patient totters or falls when the eyes are closed. It is caused by anæsthesia of the soles of the feet and in disturbance of the muscular sense in the extremities. It is a frequent symptom of *tabes*. *Paræsthesia* refers to sensations of tingling, creeping, numbness or formication.

Girdle pain (*cincture-feeling*), is a sensation of constriction around the body (usually in the region of the dorsal vertebræ), and is present in diseased conditions of the posterior columns of the cord.

Spontaneous Pain.—*Headache* (cephalagia) may be caused by: 1. Meningitis (pain violent and persistent). 2. Syphilis (nocturnal exacerbations). 3. Neurasthenia and hysteria. In the latter affections, the pain is limited to a small spot along the sagittal suture (*clavus hystericus*). 4. Migraine; paroxysms of pains occurring usually on one side of the head at irregular periods and attended with vaso-motor and gastric disturbances. 5. Intoxication from lead, alcohol, mercury and in uræmia. 6. Acute infectious diseases. 7. Anæmia. 8. Hyperæmia.

Nitrite of amyl inhalations diminish anæmic and increase hyperæmic headaches. *Infants* manifest severe headache by boring of the head into the pillow.

Neuralgia.—The chief symptom of this affection is pain; it is paroxysmal, usually unilateral, follows the course and distribution of a nerve, inflammatory symptoms are absent and tender points (*puncta dolorosa*) can be felt along the course of nerve in the intervals of pains but more pronounced during the paroxysms. The painful points are found where the nerves pass through bony canals or penetrate the fasciæ of muscles.

MOTOR SYMPTOMS OF IRRITATION.

Spasms are involuntary muscular contractions of a single muscle or a group of muscles. They may be *clonic* (alternate contraction and relaxation of a muscle) or *tonic*

(rapid recurrence of contractions, so that affected muscle appears in a fixed condition). *Convulsions* are clonic spasms extending over the entire body.

Clonic-tonic spasms occur in epilepsy, puerperal convulsions, uræmia, irritation of the cortical centers (tumors abscess, etc.), at the onset of acute febrile disease, and in children from reflex causes (dentition, intestinal worms, indigestion, etc.)

Tonic spasms occur in: tetany and tetanus. In *tetany*, "*Trousseau's sign*" is characteristic, *i. e.*, energetic contractions can be excited by pressure of the arm in the regions of the *median nerve and brachial artery.* Mechanical and electrical excitability of the peripheral nerves and muscles also exist. Temperature is normal. In *tetanus*, there is an elevation of temperature. The disease is as a rule fatal.

Varieties of Local Spasm.—Tonic spasm of the *internal rectus* is recognized by *strabismus*. *Nystagmus* is a bilateral affection frequently occurring in *multiple sclerosis* and is particularly marked when the patient looks at remote objects. It is due to clonic spasm of the muscles of the eyeballs. *Trismus* is a tonic spasm of the masticatory muscles (lock-jaw) and occurs in *tetanus*.

Facial spasm (convulsive tic) affects the muscles of the face. Partial spasm of the eyelids causes constant winking (*nictitating spasm*), if the entire *orbicularis palpebrarum* is affected the eyes may be firmly closed (*blepharospasm*). *Spasm of the Œsophagus* frequently occurs in hysteria (*globus hystericus*).

Laryngismus stridulus, is caused by spasm of the glottis and is frequent in children. *Hiccough*, is spasm of the diaphragm (*singultus*). *Writer's cramp* and similar professional neuroses consist of spasmodic contraction of definite muscle-groups when certain movements are made.

Thomsen's disease is characterized by the occurrence of spasmodic rigidity of voluntary muscles, when they are called into action after intervals of rest (*intentional spasms*).

Contracture is the permanent contraction of a muscle, which fixes the limb either in a flexed or extended position. Contracture of the muscles occurs in organic diseases of the spinal cord (especially if the lateral columns are implicated) and is associated with an increase of the spinal reflexes.

Tremor consists of slight contractions of bundles of muscular fibres. It is physiological after physical and psychical exertion. Tremor may be permanent and still physiological in old people (*tremor senilis*). *Tremor alcoholicus* occurs particularly in the extremities and tongue. In *morbus Basedowii* (exophthalmic goitre) a fine tremor also occurs. The tremor of *paralysis agitans* is controlled by active exertion and ceases during sleep. The thumb closes on the fingers as in "rolling pills."

Intentional tremor is observed in voluntary movement of the muscles and is an important symptom of *multiple sclerosis*. It is also present in *tremor mercurialis*. Trembling of the eyes (*nystagmus*) is present in multiple sclerosis, hysteria and in affections of the eye.

Fibrillary contractions of muscular bundles are seen in *atrophic paralyses*, particularly in *progressive muscular atrophy*.

Choreic movements are quick, involuntary and incoördinate movements, which prevent and interfere with voluntary motion. They cease during sleep and are pathognomonic of *chorea*. Choreic movements on one side of the body (*hemichorea*) can precede or follow hemiplegia (lesion in the posterior part of internal capsule).

Athetosis. The characteristic movements of this affection are most pronounced in the hand and fingers. The fingers are never at rest and they are constantly flexed, extended or intertwined. Athetosis may be idiopathic but more frequently it is symptomatic of *cerebral infantile paralysis*. *Hemiathetosis* has the same significance as hemichorea.

Catalepsy is characterized by a peculiar rigidity of the muscles (*flexibilitas cerea*) which enables the physician to put the limbs in any posture where they will remain fixed until the position is again changed. Catalepsy is most frequent in hysteria. Also present in meningitis and in certain psychoses (*melancholia attonita*).

REFLEXES.

Reflexes are of two kinds, *superficial* (skin) and *deep* (tendon). The former are excited by irritating the skin, the latter by exciting the tendons or fasciæ of muscles.

Skin Reflexes.—1. *Plantar reflex.* This is excited by tickling the sole of the foot. It causes a dorsal flexion of the foot and when the irritation is strong, the leg is drawn up.

2. *Cremaster reflex.* Irritating the inner side of the thigh will cause retraction of the testicle.

3. *Reflex of the abdominal walls.* Irritating the skin of the abdomen causes contraction of the abdominal muscles. Irritating the skin of the scapular (*scapular reflex*) and gluteal (*gluteal reflex*) regions causes contraction of the corresponding muscles.

Skin reflexes are *absent* or *diminished* when the *reflex circuit* (centripetal nerve, anterior horn of spinal cord and motor nerve) is broken. This occurs in affections of the peripheral nerves and spinal cord.

Tendon Reflexes.—1. *Patellar reflex* (*knee-jerk*) is obtained by percussing the patellar tendon while the leg is crossed and completely relaxed; this is followed by contraction of the quadriceps and the leg is extended. 2. *Tendo Achillis reflex:* percussing this tendon causes contraction of the calf muscles. 3. *Foot clonus;* if the leg is slightly bent upon the thigh and the foot quickly flexed rythmical contractions of the calf muscles will ensue. Tendon reflexes in the upper extremities rarely occur in health.

In health the knee-jerk is almost constant (about 2% of all adults fail to show the knee-jerk), the tendo Achillis reflex is frequent, while a persistent foot clonus never occurs. Tendon reflexes are absent when the reflex circuit is broken. The *Reflex circuit* is formed by the nerves (*sensory*) going to the spinal cord from the muscle or tendon, the motor nerves going to the muscles and by that portion of the spinal cord connecting both. The tendon reflexes are absent in peripheral paralyses, tabes dorsalis and poliomyelitis. *Increased reflexes* occur when the *inhibitory centers* are diseased or their fibres interrupted in their course from the brain through the pyramidal tracts of the spinal cord.

Paradox contraction (Westphal) occurs in multiple sclerosis and paralysis agitans. It consists of a contraction of the *tibialis anticus* with prominence of its tendon when the foot is firmly and quickly flexed, the foot remaining in this position for a few minutes after it is let go.

Reflex Functions.—1. *Pupillary reflex.* The pupil is supplied by the *oculomotorius* for the *sphincter* and the *sympathetic* for the *dilator pupillæ.* Irritation of the former nerve causes contraction (*myosis*), of the latter, dilatation of the pupil (*mydriasis*). The cen-

ter for the pupil reflex (*centrum cilio-spinale*) is situated in the lower cervical region. In *tabes*, the pupils contract to accommodation but not to light (*Argyle Robertson symptom.*) The pupils are also contracted (*myosis spinalis*) and irregular in tabes and paretic dementia.

2. Disturbances in the passing of the urine and fæces as well as the sexual reflex occur in lesions involving the lumbar region of the spinal cord.

Mucous membrane reflexes. 1. *Conjunctival:* closing the eyes when eyeball is touched. 2. *Pharyngeal:* nausea or vomiting when the pharynx is touched. 3. *Coughing* when the larynx or the air-passages are irritated. 4. *Sneezing* when the nasal mucous-membrane is irritated.

EXAMINATION OF THE NERVES AND MUSCLES BY ELECTRICITY.

The faradic and galvanic currents are employed in medical diagnosis. Static electricity (*Franklinism*) is sometimes used as a therapeutical agent.

The *faradic current* is produced by the magnetizing and demagnetizing of a bar of soft iron by means of a galvanic current. The strength of the faradic current is regulated by sliding one coil over the other as indicated by a graduated scale. The strength of the galvanic current is regulated by the number of elements used or by means of a *rheostat.* Electricity is applied to the body by means of *electrodes.*

Electro-motive Force. This is the work which a definite quantity of electricity can do and is influenced by the resistance offered to the current. The strength of electric currents is determined by *Ohm's law: The strength or intensity* (*I*) *of the current is always proportionate to the electro-motive force* (*E*) *divided by the resistance.* This is mathematically represented by the following formula: $I=\frac{E}{R}$. An *ampere* is the intensity of the current (I) generated by the electro-motive force (E) of 1 *volt* in an electric current of resistance (R) of 1 *ohm.* One volt equals $\frac{9}{10}$ the electro-motive force of a Daniell element; one ohm equals a column of mercury 106 cm. long, and 1 square millimetre in section (1.06 Siemen's unit). The resistance

of the human body is diminished by saturation of and pressure upon the electrodes. The relative resistance of the tissues is represented by the following figures (100 taken as the maximum); eye, 4; muscle, 6; nerve, 10; fat, 75; bone and skin, 100. The epidermis when dry, is practically a non-conductor of electric currents.

Measurement.—The unit of current strength adopted for medical purposes is the *milliampere* which is the one millionth part of an ampere. Galvanometers now employed are divided into milliamperes and from the deflections of a needle, the number of milliamperes can be noted.

The Poles of the Galvanic current.—The galvanic current is generated by the contact of dissimilar metals exposed to chemical action. The simplest form of a galvanic cell, consists of a plate of zinc and one of carbon immersed in a vessel containing dilute sulphuric acid. The wire connected with the zinc is the negative pole or *Kathode* (Ka) while the wire connected with the carbon is the positive pole or *Anode* (An).

One pole is distinguished from the other as follows: With a weak current place both electrodes on either cheek; a peculiar taste is experienced at the anode electrode. If the wires are dipped in water, bubbles of hydrogen accumulate about the kathode. If the wires are placed on moistened blue litmus paper the latter will become red at the anode. If the wires are immersed in a solution of iodide of potash containing starch, a blue color, due to free iodine and starch, appears at the anode. German authors employ the symbols S, for closing (*Schliessung*), O, for opening (*Oeffnung*), and Z, for contraction (*Zuckung*).

Electro-Diagnosis. — The indifferent electrode (usually large) is placed on the sternum, whereas the different or active electrode (usually small) is placed on the nerve or muscle to be examined. A muscle may be made to contract *directly* by placing the electrode on the muscle or *indirectly* by placing it on the nerve (*motor point*). Stimulation of the motor points indirectly excite the muscles to contraction, each muscle possessing a motor point which has been empirically established (Figs. 19–23).

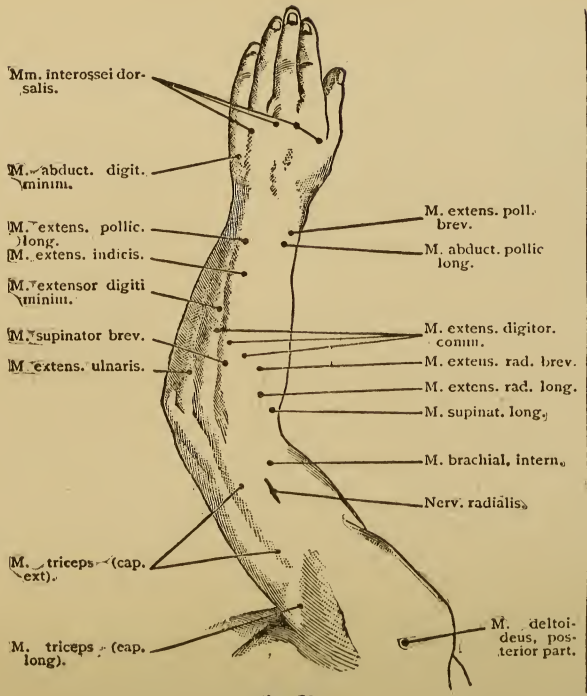
Mm. interossei dorsalis.
M. abduct. digit. minim.
M. extens. pollic. long.
M. extens. indicis.
M. extensor digiti minim.
M. supinator brev.
M. extens. ulnaris.
M. triceps (cap. ext).
M. triceps (cap. long).
M. extens. poll. brev.
M. abduct. pollic long.
M. extens. digitor. comm.
M. extens. rad. brev.
M. extens. rad. long.
M. supinat. long.
M. brachial. intern.
Nerv. radialis.
M. deltoideus, posterior part.

Fig. 20.

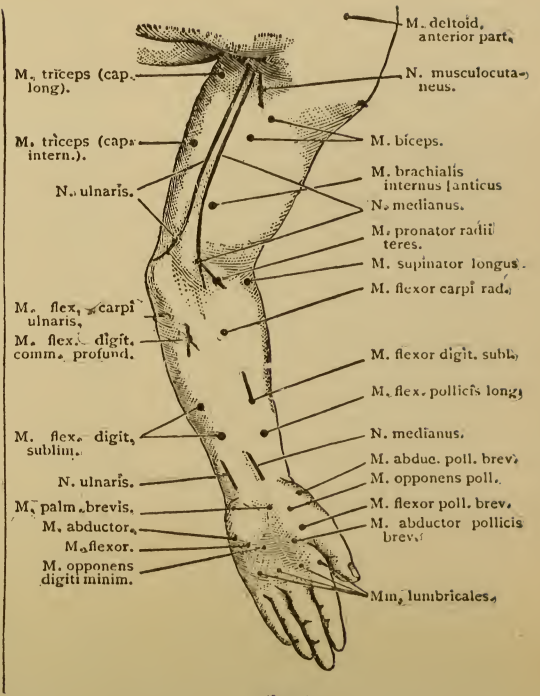
M. deltoid. anterior part.
M. triceps (cap. long).
N. musculocutaneus.
M. triceps (cap. intern.).
M. biceps.
N. ulnaris.
M. brachialis internus [anticus
N. medianus.
M. pronator radii teres.
M. supinator longus.
M. flexor carpi rad.
M. flex. carpi ulnaris.
M. flex. digit. comm. profund.
M. flexor digit. sublim.
M. flex. pollicis long.
M. flex. digit. sublim.
N. medianus.
N. ulnaris.
M. abduc. poll. brev.
M. opponens poll.
M. palm. brevis.
M. flexor poll. brev.
M. abductor.
M. abductor pollicis brev.
M. flexor.
M. opponens digiti minim.
Mm. lumbricales.

Fig. 19.

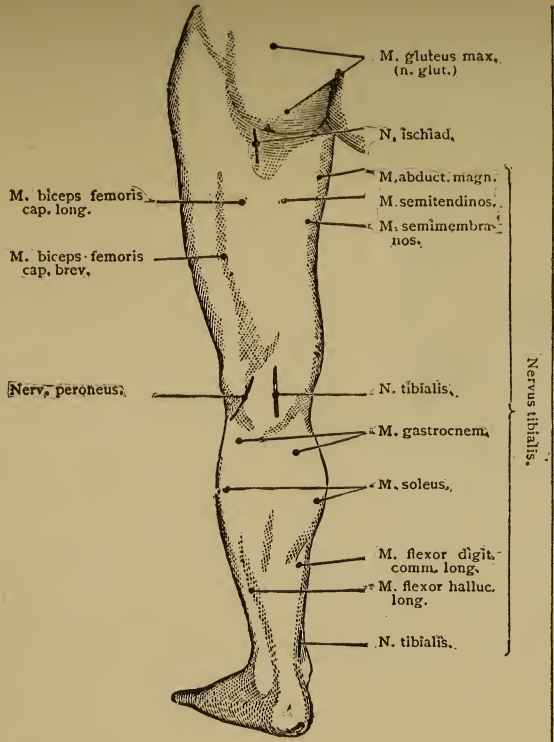
M. gluteus max. (n. glut.)
N. ischiad.
M. abduct. magn.
M. semitendinos.
M. semimembranos.
M. biceps femoris cap. long.
M. biceps femoris cap. brev.
Nerv. peroneus.
N. tibialis.
M. gastrocnem.
M. soleus.
M. flexor digit. comm. long.
M. flexor halluc. long.
N. tibialis.
Nervus tibialis.

Fig. 22.

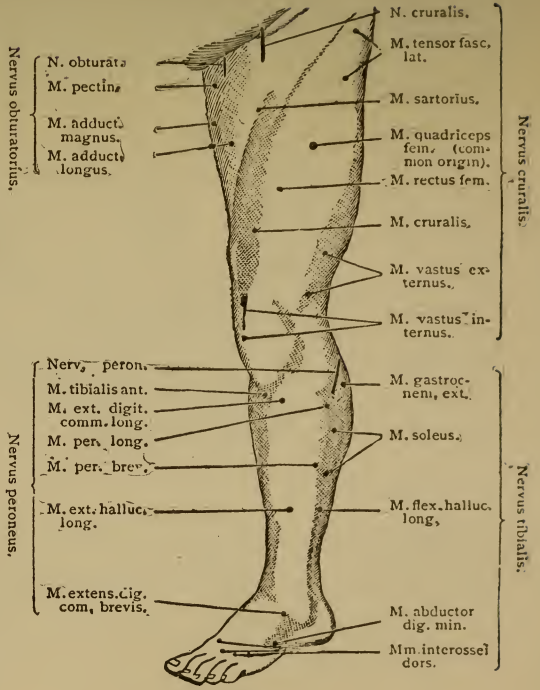
N. cruralis.
M. tensor fasc. lat.
M. sartorius.
M. quadriceps fem. (common origin).
M. rectus fem.
M. cruralis.
M. vastus externus.
M. vastus internus.
Nervus cruralis.
Nervus obturatorius.
N. obturat.
M. pectin.
M. adduct. magnus.
M. adduct. longus.
Nerv. peron.
M. tibialis ant.
M. ext. digit. comm. long.
M. per. long.
M. per. brev.
M. ext. halluc. long.
M. extens. dig. com. brevis.
Nervus peroneus.
M. gastrocnem. ext.
M. soleus.
M. flex. halluc. long.
M. abductor dig. min.
Mm. interossei dors.
Nervus tibialis.

Fig. 21.

With the *faradic current* muscular contractions occur by *direct* or *indirect irritation.* The weakest current necessary to produce muscular contraction is noted by the position of the coils as indicated by a scale in millimetres.

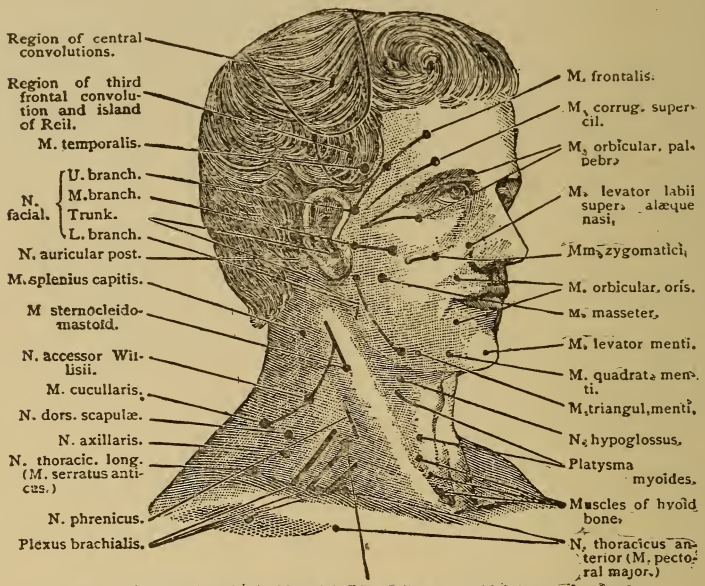

Fig. 23.

With the *galvanic current*, the *Kathode* is placed on the muscle or nerve to be examined. The strength of current is gradually increased until a contraction occurs at the closing of the current (*Kathodal closing contraction, KCC.*) The intensity of the current necessary to produce contraction is indicated by the galvanometer or by the number of elements used.

KCC occurs normally in superficial motor nerves with a current intensity of 1–3 milliamperes (*MA*).

By means of the polarity changer or commutator, without changing the position of the electrodes the kathode may be converted into the anode and *vice versa.*

Formula of Normal Polar Action.

1. *Weak current:* Kathodal closing contraction (*KCC*).

2. *Medium current:* Strong Kathodal closing contraction (KCC), moderate anodal closing contraction (*ACC*) and moderate anodal opening contraction (*AOC*).

3. *Strong current:* Tetanic Kathodal closing contraction (*Te KCC*), strong anodal closing contraction (*ACC*), strong anodal opening contraction (*AOC*) and Kathodal opening contraction (*KOC*).

This formula only holds good when the muscles are *indirectly* stimulated with the galvanic current. In *direct* stimulation of the muscles there are only closing contractions, and *ACC* may be equal to *KCC*.

Quantitative changes of electrical irritability refer to the energy and amplitude of contractions. The electrical irritability is *increased* in tetany; *diminished*, in paralysis resulting in simple atrophy as in apoplexy and in bulbar and spinal paralyses when the trophic ganglia are intact.

Increased Electric Excitability occurs when a weak current will produce energetic contractions and it is *diminished* when a strong current is necessary to excite muscular contraction. In unilateral paralysis the healthy side may be used as a basis of comparison.

Qualitative changes refer to abnormal polar reaction. If a motor nerve is cut off from its trophic centre or if the trophic centre is diseased, paralysis occurs and the nerve degenerates *(degenerative atrophy)*, the degeneration extending to the muscle supplied by it. There is also diminished electric irritability of the nerve for the faradic and galvanic currents and after 8 to 14 days the irritability is entirely lost. Direct faradic irritability of the muscle is also entirely lost. After 2 weeks direct galvanic irritability of the muscle is increased and contractions occur even with mild currents; the contractions are not short and quick as in health, but long and slow. *ACC* with the same current, occurs as soon or sooner than *KCC; KOC* may be obtained with weaker currents than the *AOC*. The foregoing reaction is *the reaction of degeneration (RD)*. In severe cases, showing *RD*, the galvanic irritability is finally lost after 4 to 8 weeks.

In curable cases showing *RD*, the voluntary power of the affected muscle is restored sooner than the electric irritability. *The reaction of degeneration* is *present* in all peripheral lesions of the

motor nerves whether of traumatic, rheumatic, toxic or diphtheritic origin; also in diseases of the gray matter of the anterior horns of spinal cord and gray nuclei of medulla. The *RD* is *absent* in all cerebral and spinal paralyses, the cause of which is central from the trophic centre.

The presence or absence of the *RD* is of value in *prognosis*. If present, it indicates either that the atrophy of the affected muscles is incurable or that at least 2–3 months will be necessary for their restoration. If absent, it indicates the absence of gross anatomical lesions and cure may result in a few weeks.

Partial Reaction of Degeneration.—In this condition the nerve retains its faradic and galvanic irritability and the muscle its faradic irritability, but the direct galvanic irritability of the muscle is increased, the normal formula of polar action is altered and the contractions are slow and long. This reaction shows anatomical changes in the muscle but not in the nerve.

INSTANTANEOUS DIAGNOSIS IN NERVOUS DISEASES *(Erb)*.

Tabes dorsalis is, as a rule, easy to diagnose; although, even at the present time, it is mistaken for *myelitis*, *spastic paralysis*, *neuritis*, etc. The diagnosis of this affection can at once be made, when the patient enters the room with an *ataxic gait*, when he complains of *lancinating pains*, *double vision*, weakness, tired feeling, and *paresthesiæ* of the legs, vesical and genital weakness. An examination of the eyes will show *contraction of the pupils*, and a failure of the latter to respond to light. The patient will totter with closed eyes, and the *knee-jerk* is *abolished*. Parkinson's disease, or *paralysis agitans*, is also capable of immediate diagnosis. The marked *inclination of the body forwards*, the *attitude of the fingers*, as if employed in holding the pen, the *immobility of facial expression*, the *peculiar gait*, as if the patient were about to tumble forward, all give, when combined with the peculiar *tremor*, a characteristic picture. The tremor is not always essential in making the diagnosis, for there are cases in which this symptom is permanently absent. Tetany.—A young man or woman is troubled with paroxysms of *tonic muscular spasms* in the hands or legs. If, now, with the end of the finger, or a lead pencil, a vertical stroke is made running from the temporal region to the lower jaw, and a rapid contraction of the facial group of muscles occurs, the diagnosis of *tetany* can with certainty be made. The diagnosis is confirmed when, after the nerve trunks of the arm are struck, violent contractions of the muscles ensue; or when, by application of the galvanic current to the nerve trunk, kathodal closure tetanus and anodal closure tetanus are rapidly developed with

even weak currents, the muscles showing increased irritability Thomsen's disease.—A young person complains with reference to a weakness and stiffness of intended movements. You ask the patient to grasp your hand, and he will be unable, when told, to immediately loosen his grasp. Now strike the exposed deltoid muscle, or the biceps, and an energetic contraction, lasting many seconds, occurs. This is *Thomsen's disease*, or *myotonia congenita*. If the symptomatic picture is to be completed, use the faradic and galvanic currents. The nerves show normal irritability; the muscles, on the contrary, are irritable and qualitatively changed *(myotonic reaction)*. Besides the tendency of the muscles to tonic spasm during attempts at voluntary movement, and the myotonic reaction, absolutely nothing else abnormal is found, other than the disproportion existing between the well-developed muscles and the weakness of the patient. Basedow's disease (*exophthalmic goitre*): The well-known symptoms, *exophthalmos*, *pulsating struma*, and *cardiac palpitations*, are so characteristic that the affection is at once recognized. Cases exist, however, where the exophthalmos is absent, or the thyroid gland not enlarged. In such cases the cardiac palpitations and acceleration of the pulse (120 to 160 beats per minute) without manifest cardiac change, auscultation of the pulsating struma, the feeling of weakness and *tremor*, the tendency to sweating, sleeplessness, and *diminished electrical conduction resistance of the skin*, are symptoms of value in undeveloped forms of Basedow's disease.

Dystrophia Muscularis Progressiva.—The patient is a child with weakness of the legs, clumsiness in walking and difficulty in rising from the recumbent or sitting position; you ask to have the clothes removed and proceed to examine the patient more carefully. A *waddling movement* is noticeable, together with *lordosis* in the lumbar region. The thighs are characteristically attenuated when contrasted with the muscles of the calf, which are increased in size. When the child is raised from the arms, the shoulders ascend to the ears and the head sinks between the shoulders, so that difficulty is experienced in lifting it in this manner, so readily accomplished in healthy children. The failure is attributed to the inability to fix the shoulders below. The individual forms of this disease can at once be recognized. The *pseudohypertrophic form*, by the increase in size of the muscles, which is not proportionate to their motor power; *the juvenile*, by the more severe involvement of the upper half of the body, the pronounced atrophy and relatively late development of the affection; the *infantile*, by the early invasion of the face. Infantile paralysis (*poliomyelitis anterior acuta*). In these cases the child presents an *atrophic paralysis* of one or more extremities, whereas individual muscles, or muscle groups, are exempt. The sphincters are intact, and the affection has occurred suddenly. Sensation is not involved, and the tendon reflexes in the affected muscle regions are absent. The examination is completed when the reaction of degeneration is determined. Multiple sclerosis: *Intentional tremor* of the hand, shaking of the entire body when an attempt is made to rise or

walk, *scanning speech*, *nystagmus*, *spastic paresis* of the legs, and history of headache, vertigo, vesical weakness, etc., constitute the characteristic symptoms of this disease. **Progressive bulbar paralysis:** Difficulty in articulation, nasal voice, thin lips and with difficulty moved, the inability to protrude an atrophic tongue in which fibrillary contractions are observed, difficulty in swallowing, etc. **Amyotrophic lateral sclerosis:** This affection shows a combination of *atrophic paresis* with *increased tendon reflexes* in the upper, and *spastic paresis* in the lower extremities.—*Deutsche Medicin. Wochenschrift*, October 17, 1889.

MEDICAL OPHTHALMOLOGY.

Ophthalmic Examination of the eye is of great importance in medical diagnosis. In the following affections the ophthalmoscope establishes or corroborates a diagnosis.

Aortic Insufficiency.—An alternate flushing and pallor of the optic disc analogous to the capillary pulse, and pulsation of the retinal vessels is observed.

Congenital Heart Lesions.—The retinal vessels are distended.

Leucæmia.—Diffuse retinitis with hæmorrhages and yellow color of the eye ground.

Pernicious Anæmia.—Œdema of the retina with hæmorrhages.

Hæmorrhage.—Loss of blood may cause impaired vision from transient anæmia of the retina or cerebral centres. Ophthalmoscopic examination not diagnostic.

Bright's Disease.—Œdema of the disc and surrounding retina with irregular white splotches, and striated hæmorrhages. About 23% of patients with renal diseases have disorders of vision at some period of the disease. The eye lesion may be the first symptom of the renal affection.

Tuberculosis.—Usually in children tubercles are deposited in the choroid, intra-ocular end of the optic nerve, retina or iris. Their favorite seat is the macular region and its vicinity. The deposit of tubercles may precede other symptoms. According to Cohnheim in all cases of *acute miliary tuberculosis*, tubercles are to be found in the *choroid;* although this view is vigorously disputed by other observers.

Diabetes Mellitus.—*Diabetic neuroretinitis* and atrophy. The retinal changes resemble those due to albuminuria. Cataract is also found and grape sugar may be detected in such lenses by chemical examination.

Tabes Dorsalis.—Atrophy of the optic nerve is an early and frequent symptom, and may precede by many years, the development of spinal symptoms. In the early stages, the discs are of a dull, reddish-gray tint, which gradually becomes paler, and at last white. Later the nerves assume a greenish tint, the surface of

the disc becomes excavated, and the retinal blood-vessels shrink. *Atrophy of the optic nerve* is also observed in *disseminated sclerosis and paralytic dementia.*

In tabes the field of vision is narrowed and the sense of color is disturbed; green being lost first, then red, and finally, yellow and blue.

Increased intracranial pressure—(*Tumors, meningitis, hydrocephalus*). The optic nerve is swollen and projects above the level of the surrounding retina; the margin of the disc is either obscured or entirely lost; the arteries are small, the veins large and tortuous (*choked disc*). These changes usually occur on both sides alike, and of equal intensity. Like changes in the fundus occur in about ¾ of all tumors of the brain.

Exophthalmic Goitre.—Dilatation and tortuosity of the retinal veins. Functions of the retina unimpaired. Spontaneous pulsation of the retinal vessels has been observed. Protrusion of the eye ball is often combined with a want of agreement between the movement of the lid and the raising or lowering of the eye (*symptom of Graefe*).

Syphilis.—(*Syphilitic retinitis*). Retina is covered by a gray film, and in the macular region numerous irregularly placed punctate spots are present.

Lead intoxication may be followed by optic neuritis with consecutive optic nerve atrophy. In *quinine poisoning*, there is pallor of the optic disc, and narrowing of the field of vision. *Alcohol and tobacco intoxication* also lead to atrophy of the optic nerve.

CHAPTER XIII.

PARASITES.

ANIMAL PARASITES.

The diagnosis of *animal parasites* is only possible in many cases after careful examination of fresh fæcal matter with the microscope. The *amœba* and *infusoria* lose their activity soon after evacuation of the stool, and for this reason Eichhorst recommends the removal of the mucus and fæces from the rectum by means of a glass tube, and direct transference of the same to the object glass. The microscope will also aid in the diagnosis of *eggs* of the intestinal parasites.

Animal parasites belong either to the *Protozoa* or to the worms. The protozoa are divided into *rhizpoda* and *infusoria*, and the *worms* into flat (*platodes*) and round worms (*nematodes*). The following parasites are of practical importance: I. Protozoa: a. *Rhizpoda*: amœba coli. b. *Infusoria*: 1. Cercomonas intestinalis. 2. Trichomonas intestinalis. 3. Balantidium coli.

II. Worms.—a. *Flat worms* (*platodes*).

1. Tænia solium.
2. Tænia saginata or tænia mediocanellata.
3. Bothriocephalus latus.
4. Tænia echinococcus.

b. *Round worms* (*nematodes.*)

1. Ascaris lumbricoides.
2. Oxyuris vermicularis.
3. Trichocephalus dispar.
4. Anchylostomum duodenale.
5. Trichina spiralis.
6. Filaria sanguinis.

Only finding in the stools the parasites, segments of the tapeworms or eggs by the microscope can be considered as diagnostic.

Protozoa. *Amœba coli.* A round granular structure with a nucleus and several vacuoles.

Cercomonas intestinalis. This parasite is pear-shaped with ciliated extremities and is $\frac{1}{30}$–$\frac{1}{25}$ of an inch long.

Trichomonas intestinalis. Almond-shaped with ciliated extremities and is $\frac{1}{25}$–$\frac{1}{10}$ of an inch long.

Balantidium coli. Pear-shaped, ciliated and with an inverted mouth. It is about ¼ of an inch long.

Tape or Flat Worms (*platodes*).—They are acquired by the ingestion of raw or insufficiently cooked meat containing the eggs. *Tænia solium* is acquired by eating ill-cooked pork.

Tænia saginata is derived from beef and the *Bothriocephalus latus*, from fish. The three species can be diagnosed by voiding of the segments (*proglottides*). The bothriocephalus is the most easily removed by medication, whereas the *T. saginata* requires the most active treatment.

Fig. 24.

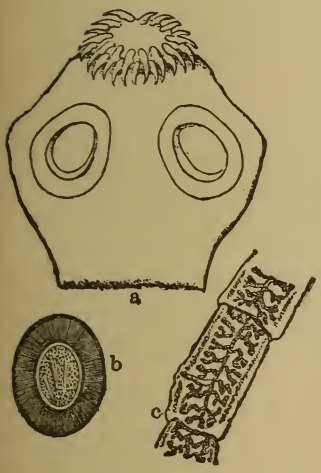

Fig. 25.

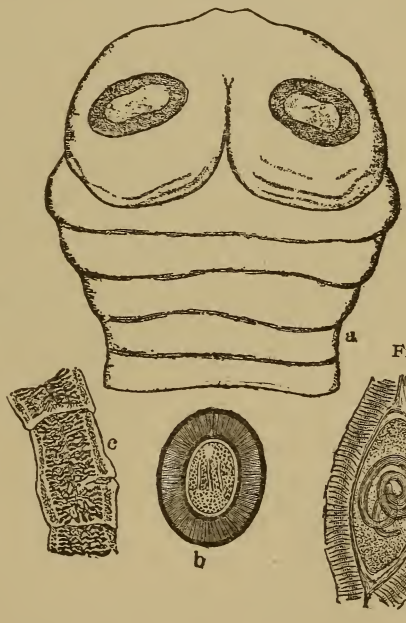

Fig. 26.

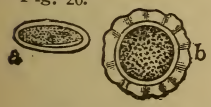

Fig. 27.

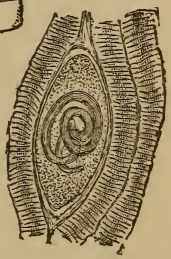

Fig. 24. Tænia solium. a. head, b. egg, c. segment.
Fig. 25. Tænia saginata. a. head, b. egg, c. segment.
Fig. 26. a. Egg of the oxyuris vermicularis. b. egg of ascaris lumbricoides. Fig. 27. Muscle trichina.

The tapeworms are composed of a head (*scolex*) and segments (*proglottides*). The eggs come from the matured segments (*hermaphrodite*), and, when taken into the stomach of an animal, the

capsules are dissolved and the embryo is dislodged. It then reaches the tissues and develops into a *cysticercus*. This cysticercus when contained in the food ingested by man develops into a new tapeworm. The method of examining a segment is to press it slightly between two object glasses and examine with transmitted light.

Tænia solium. This (tapeworm) is 1–3 meters (yards) long. The head is about the size of a pin's head, containing 4 suckers, and is prolongated into a proboscis (*rostellum*) surrounded by a double row of hooks (20–30). The matured proglottides have the sexual opening at the side somewhat behind the middle. They have a uterus with 7–10 thick lateral branches, which subdivide. The eggs are round or oval with a striped shell containing an embryo with 6 hooks. This cystic condition of the tænia (*cysticercus cellulosæ*) may be found under the skin, in the brain, eye, and muscles, causing severe disturbances.

Tænia saginata. (7–8 meters long). Larger than the former. The head has 4 suckers without rostellum or hooks. Sexual openings are on the sides of the proglottides. The uterus has 20–30 lateral branches, which are finer than those of the *T. solium.* The eggs are like the latter but larger. The cysticercus does not develop in the tissues of man.

Bothriocephalus latus (4–5 meters long.) Lancet-shaped head with two lateral grooves. Matured proglottides are broader than long and genital apertures are found in the median line.

The uterus is arranged in the form of a rosette around the sexual openings.

Tænia echinococcus. Found in the dog. It has a head (*scolex*) with hooklets (30–40), suckers and 3 segments, of which the last one alone is matured. Only the cystic form or *hydatid* is found in man, which he acquires by eggs introduced into the alimentary canal. When the embryo migrates from the intestine to some other organ (liver, spleen, kidneys, lungs, etc.), it is transformed into a cyst incapable of active motion.

The *hydatid cyst* consists of an external elastic cuticle, and an internal lining, the parenchymatous layer. An echinococcus cyst may be simple (*unilocular*), filled with daughter cysts, or it may contain a large number of minute cavities filled with a gelatinous substance and with concentrically arranged walls (*multilocular*).

The nature of an echinococcus cyst can only be determined by puncture and examination of the withdrawn fluid. The fluid of a cyst is usually clear, neutral or alkaline, with a specific gravity of 1008–1013. It contains little or no albumen, but large quantities of sodium chloride, frequently grape sugar and *succinic acid*. The latter is detected as follows: Evaporate the fluid and add hydrochloric acid; shake with ether and allow the ether to evaporate. If succinic acid is present it remains as a crystalline mass. The latter when dissolved in water will give with chloride of iron, a rust-colored gelatinous precipitate of succinate of iron. Heated in a test-tube irritating fumes of the succinic acid are given off, causing cough. The microscopic demonstration of the echinococcus is based on the presence of scolices or their hooklets, or the cystic membrane. The scolices are easily recognized by the presence of suckers and hooklets. The hooklets, are small and transparent and a magnifying power of 500 diameters is necessary for their detection.

In the case of *acephalo-cysts, i. e.*, non-propagating cysts, examination of the membranes is necessary in diagnosis. The membranes have a characteristic striped appearance.

Round Worms.—*Ascaris Lumbricoides* (*round worm*). Its habitat is in the small intestine, but it may pass to other parts of the body. It resembles the ground-worm in shape. The male is smaller (4–6 inches) than the female. When the worm does not pass off with the fæces, then examination must be made for the eggs, which are expelled in large quantities. They have a thick, concentrically striped shell, upon which lies a thick, tuberculated albuminous cover. These worms are generally innocuous.

Oxyuris vermicularis (*thread worm*). Its habitat is chiefly the rectum. When passed in a living condition, it exhibits a lively motion. These worms cause intense itching in the region of the anus. The female is longer ($\frac{1}{2}$ inch) than the male ($\frac{1}{8}$–$\frac{1}{4}$ inch). The female has a finely pointed tail-end, whereas the male (more rare) has a blunt tail-end curved in the shape of a hook. The diagnosis is made from the constant passage of the worms with the stool, or by searching for the eggs which are oval and possess a thin shell.

Tricho-cephalus dispar (*whip-worm*). It inhabits the cæcum, although rarely in large numbers. It is 1½–2 inches long, and has a thread-like head extremity, while the tail-end is considerably thicker. The eggs (*necessary for diagnosis*) are about the size of those of the oxyuris and shaped like a lemon.

Anchylostomum duodenale (*hook-worm*). It inhabits the small intestine, where it hooks itself fast and sucks blood, causing pronounced anæmia. This worm is found in tunnel workmen, brickmakers and miners. In persistent anæmia of these individuals the worm should be sought for after the administration of an anthelmintic.

The anchylostomum is somewhat larger then the oxyuris and its mouth is armed with three pairs of hooked teeth. The *eggs* are about the size of those of the oxyuris and are usually found in the stages of vitelline segmentation. The eggs are developed a few days after the passage of the larvæ from the intestines. If the eggs are not recognized, the fæces are allowed to stand in a warm place for 2 or 3 days and then reëxamined with the microscope.

Trichina spiralis. Found in the human body as *muscle* and *intestinal trichinæ.* It enters the intestine by means of trichinosed pork.

When the pork containing the live encysted trichinæ reaches the stomach the capsules are dissolved and the trichinæ set free. They *mature* in the intestine in about 2½ days, and bring forth after 5–7 days young trichinæ which *migrate* through the intestinal walls and reach the striated muscles where they become encapsuled. A single worm may bring forth 1000–1300 young. The *male trichina* is $\frac{1}{18}$ inch long, the female $\frac{1}{7}$ inch long. *Trichinosis* is attended with various symptoms. When trichinous flesh is first ingested, there are symptoms of gastric and intestinal catarrh.

Muscular invasion is attended with fever, œdema, partial paralyses, muscular abscesses and pain. The symptoms reach their height in the 4th or 5th week. The violence and gravity of the symptoms depend generally on the number of trichinæ which have entered the muscles. The trichinæ are most abundant in the diaphragm, intercostal, cervical and laryngeal muscles. They are less often found in the muscles of the extremities where they are usually crowded together at the attachment of the muscle to its tendon.

The removal of a piece of muscle for microscopical examination is often necessary for diagnosis during life. An accurate and simple method for determining the presence of trichinæ in pork is the following: The suspected piece of meat is put into a mixture of pepsin and dilute hydrochloric acid and allowed to remain in a conical-shaped glass at the body temperature. In the latter after digestion the free trichinæ are deposited and may be removed by means of a pipette for microscopical examination.

Filaria sanguinis hominis. Indigenous only within the tropics (Egypt. Brazil, India, etc.). In its sexually mature form it is a filiform worm (8–10 ctm. long). It inhabits the lymphatics especially those of the scrotum and lower

limbs. It obstructs the lymph-vessels and causes inflammations which terminate in elephantiasis of the tissues with œdema and lymphangiectasis.

The embryos pass from the lymphatics into the blood and cause *hæmaturia* and *chyluria*. The embryos may be excreted with the urine. An examination of the blood with the microscope shows the presence of embryos which appear as little worms $\frac{1}{150}$ inch long and as broad as the diameter of a red blood corpuscle. They are found in the blood at times only during the *night hours*, hence an examination at this time may be necessary. They may remain for months or years in the body without creating manifest disturbances.

EXTERNAL PARASITES.

The external parasites may be divided into animal and vegetable.

ANIMAL PARASITES.

Sarcoptes hominis (acarus scabiei).—The itch mite bores its way obliquely through the horny layer of the skin until it reaches the rete mucosum in the neighborhood of the papilla. In this way it forms furrows *(cuniculi)* at the ends of which the insect is found. In its course it leaves behind excreta (yellow, brown or black grains and lumps). The female lays its eggs in the furrow and as these are hatched (8–14 days), the young mites may be seen in all stages of development.

The itch mite selects the soft skin between the fingers, flexor surfaces of the joints and parts pressed upon by tight portions of clothing. In males, the penis and lower part of the abdomen are usually invaded. The parasite is secured by passing a needle along the furrow toward the papule; on tearing open the furrow, the acarus usually adheres to the point of the needle. The best specimen is obtained by snipping by means of curved scissors, a piece of the skin containing the furrow.

Acarus folliculorum.—Occurs in about 10 per cent. of all healthy adults in the sebaceous and hair follicles of the face.

Pediculus capitis (head louse).—Inhabits the hairy scalp. It fastens its eggs to the hairs by means of a chitinous covering and they may be seen as grayish oval bodies. The young louse emerges in about 8 days.

Pediculus pubis (crab louse). — Smaller than the former and invades the hairy parts of the genitals.

Pediculus vestimentorum (body louse).—Inhabits the underclothing and passes to the surface of the body in order to feed.

Pulex irritans (flea).—This draws blood from the skin like the pediculi. A small punctiform hæmorrhage surrounded by a reddened areola is found at the point attacked.

Dracunculus medinensis (Guinea worm).—Found in those residing in the tropics. Occupies the subcutaneous tissue about the ankle. The usual method of securing the parasite is by fixing an exposed portion to a short rod and twisting it a little every day, until the entire worm is withdrawn without breaking.

Chigoe (pulex penetrans).—The sand-flea is principally confined to the West Indies and attacks the bare feet of the natives.

VEGETABLE PARASITES.

Mould and yeast fungi, like bacteria, draw their nutriment from organic carbon compounds. The mould fungi are seen in decaying organic substances, while the yeast fungi *(blastomycetes)* set up alcoholic fermentation. *Morphology.*—As they occur in man, the mould fungi appear in the form of jointed or unjointed filaments *(hyphæ)* and of ovoid or spherical cells. The filaments form compact masses *(mycelia)*. The ovoid or spherical cells are the spores *(conidia)*.

Achorion Schœnleinii (*favus fungus*).—Present in *favus*, a skin disease appearing on the hairy parts of the scalp and characterized by dry, flat, yellow crusts, circular and depressed in the centre, through which the hairs project. If a small piece of a crust is placed on a glass with a little caustic potash and examined with a ¼-inch objective, spores and a densely felted mycelium may be seen.

Trichophyton Tonsurans.—This is the fungus of *herpes tonsurans* and *parasitic sycosis*. The fungus filaments are present in the epidermis, the spores in the hair. Both fungi give characteristic cultures, which by inoculation reproduce their respective diseases.

Microsporon Furfur (*Pityriasis versicolor*).—The yellowish epidermic scales produced in this affection may be confounded with pigmented spots. The fungus is demonstrated by adding to a few of the scraped off scales on an object glass, a few drops of caustic potash. Under the microscope, a number of branched filaments (*mycelium*) and shining spores (*conidia*) are seen. The same procedure is applicable in examining for other fungi in the hairs and scales of the skin.

Oidium Albicans.—This fungus forms the white patches known as *thrush* (or *aphthæ*) present in the mouth, pharynx, œsophagus and stomach of weakly children and debilitated persons. It consists of branching filaments with shining spores at the points of bifurcation.

Aspergillus Glaucus and Niger.—Found in the external auditory meatus and nose. They may also vegetate in the lungs (*pneumonomycosis aspergillina*) where they are usually secondary to destructive processes (gangrene, tuberculous cavities, etc.). They form double-contoured, branched filaments containing pigmented spores.

BACTERIA—(Schizomycetes).

Morphology and Physiology.—These parasites are minute vegetable organisms of the lowest and simplest form and are widely distributed in the air, water, surface soil and about substances, animal and vegetable, undergoing decay. In their growth, the bacteria develop certain products *(ptomaines)* which, if poisonous, are called *toxines*. It appears at present, as if the deleterious effects of bacteria are largely due to ptomaines. Brieger has shown that the ptomaines cadaverin, neurin and mytilotoxin will produce in rabbits convulsions, paralysis, gastro-intestinal disturbances, etc. The ptomaine of tetanus *(tetanin)* will produce tetanus by inoculation. Coloring matters are often developed by bacteria. Bacteria are always present in the cavities of men and animals and are only active in the presence of moisture and may remain inert for a long time, either as spores or developed organisms to become again active under favorable conditions. At a temperature below 23°F., they are incapable of proliferation. They are most active at about the body temperature. In fluids all bacteria are killed when the boiling point of water is reached (212°F.) When dry, they are more resistant to heat than when moist. The spores are more resistant than the bacteria. Of all the agents which reduce the activity or destroy the bacteria, *corrosive sublimate*, even in extremely dilute solutions, is the most powerful. An aqueous solution of 1:20,000 kills the spores of bacilli in ten minutes; and a solution of 1:300,000 stops the germination of bacterial spores. Disinfecting agents should be used in aqueous solution. In alcohol or oil, their action

is feeble. Bacteria may be *pathogenic* or *non-pathogenic*. The former multiply in the living organism and are the cause of infectious diseases. The non-pathogenic bacteria only vegetate on dead organic material *(saprophytes)* and are the cause of putrefaction and fermentation.

Phagocytes.—This term has been applied to certain cells of the body which are capable of taking up into their protoplasm and destroying or digesting bacteria which get into the tissues. The resistance of the organism to certain infectious diseases may be explained by the theory of *phagocytosis*.

Classification of Bacteria.—The following classification is based on the shape of the various known species and will no doubt be modified according to our increased knowledge of these organisms.

1. *Spheroidal bacteria (micrococci).*—They may occur in beaded chains *(streptococci)*, in pairs *(diplococci)*, in masses *(zooglea)*, or in grape-like groups *(staphylococci)*. *Pathogenic micrococci* have been found in the following diseases: diphtheria, scarlatina, ulcerative endocarditis, erysipelas, cerebro-spinal meningitis, pneumonia, osteo-myelitis, periositis, pyæmia, puerperal fever, gonorrhœa, and in connection with suppurative inflammation.

2. *Rod-shaped bacteria (bacilli).*—This form of bacterium may form long, slender, filiform bacilli *(leptothrix)*. *Pathogenic bacilli* have been found in: malignant pustule, tuberculosis, typhoid fever, leprosy, Asiatic cholera and glanders.

3. *Spiral-shaped bacteria.*—The most important species of this group is the *spirillum* of relapsing fever.

METHOD OF DEMONSTRATING THE BACTERIA.

For clinical purposes the coloring of micro-organisms in a dried, cover-glass preparation is generally employed, but in a few instances this procedure will not suffice, necessitating artificial cultivation of bacteria and inoculation into healthy animals.

METHOD OF MAKING DRIED COVER GLASS PREPARATIONS.

Spread with a sterilized needle (sterilized by heating in a flame and allowing to cool) the fluid to be examined on a clean cover-

glass. With another cover-glass rub both together so that a thin film of the fluid is deposited on each. The cover-glasses are next dried by gentle heating over a flame which fixes and renders insoluble any albuminous matter mixed with the bacteria. The next step is staining of the preparation.

Staining.*—Any one of the following basic aniline colors, either in concentrated alcoholic or watery solution after filtration, may be employed: *Fuchsin* (*muriate of rosaniline*), methyl blue, methyl-violet, gentian-violet, Bismarck brown (*vesuvin*) or malachite. To the cover-glass preparation a few drops of any of the above solutions may be added and the staining is usually complete in two or three minutes. The cover-glass is next washed in water and placed on a slide when it is ready for the microscope. If the specimen is to be a permanent one, then the cover-glass is allowed to dry after washing in water when it is mounted in balsam. Balsam softened with oil of cedar or xylol is usually used, because chloroform will decolorize the bacteria.

Nearly all the micro-organisms may be shown with any of the basic aniline colors except the tubercle bacillus (*see page* 65).

To stain the *gonococci* (*Neisser's method*) press a drop of gonorrhœal pus between two cover-glasses, after which they are drawn apart, allowed to dry and then stained with methyl blue. According to Steinschneider, the application of the Gram method of staining is necessary to the satisfactory study of the gonococcus inasmuch as the only positive characteristic of this coccus is, that it is not stained by this method, while nearly all other diplococci, found in the urethra are colored thereby. The *bacilli of syphilis* are conveniently stained according to the method of Giacomi. The cover-glass preparations are stained for a few minutes in a heated solution of fuchsin in aniline water, then washed in water containing a few drops of a solution of chloride of iron; washed, and then decolorized in a concentrated solution of chloride of iron. The syphilis bacilli are colored red while all other bacteria are decolorized.

Gram's Method.—For isolated staining of the bacteria, this method is to be recommended.

Solution 1. A saturated solution of gentian violet in aniline water.

Solution 2. Solution of iodine in iodide of potash (iodine, 15 grains, iodide of potash, 30 grains, distilled water, 9½ ounces).

Solution 3. Absolute alcohol.

Solution 4. Saturated watery solution of Bismarck brown.

The cover-glass preparation is placed in solution 1, for three minutes; then in solution 2, for about two minutes; then in solution 3, until the preparation is decolorized. The micro-organisms are now stained a bluish black; but in order to make the colored

*All the materials, instruments, microscopes, etc., needed for bacteriological study, can be obtained at the opticians, Henry Kahn & Co., 642 Market street, and Hirsch, Kahn & Co., 333 Kearny street, San Francisco.

microbes more evident, solution 4, is used for back-ground staining. The preparation may be examined in water or dried and examined in balsam.

Fig. 28.

a. Streptococcus erysipelatis. b. Bacillus tuberculosis. c. Bacillus lepræ. d. Gonococcus. e. Pneumococcus. f. Bacillus typhosus. g. Bacillus anthracis. h. Spirillum of recurrent fever. i. Comma-bacillus of Asiatic cholera.

In examining stained bacteria, good homogeneous immersion lenses (not less than $\frac{1}{12}$) and an achromatic condenser of approved pattern, are indispensable. The condenser is best used without the diaphragm.

APPENDIX.

THE NERVOUS SYSTEM.

EXAMINATION OF THE MIND.

Disturbances of Consciousness.—*Stupor,* sleepiness from which the patient is easily awakened. *Sopor,* sleepiness from which the patient is aroused with difficulty. *Coma,* complete loss of consciousness from which the patient cannot be aroused.

Coma may result from *traumatism, organic disease of the brain, epilepsy, diabetes, sunstroke, uræmia, infectious diseases, hysteria, carcinoma,* and from drugs (*alcohol* and *opium*). The following points are of value in differentiating coma. Evidences of paralysis and conjugate deviation of the eyes speak for *apoplexy;* albumen and casts in the urine, and convulsions and vomiting at the beginning of coma, speak for *uræmia;* in *opium poisoning,* the contracted pupils and slow respiration, are characteristic.

Delirium is a wandering of the mind, manifested by a rapid flight of ill-assorted thoughts, which are unintelligible. It may result from *inanition, alcoholism, uræmia, infectious diseases,* and *hysteria.*

Acute Delirium (*Bell's Mania*) is a disease running a rapidly fatal course. Its cause is unknown, as there are no definite post-mortem lesions. In delirium associated with insanity, the patient is usually in good health in all save his intellect.

Vertigo (*dizziness*) is a temporary loss of consciousness. It is symptomatic of a large number of conditions. It may only indicate a sense of unstable equilibrium, as occurs in *diplopia.* Vertigo may result from: *Congestion* or *anæmia of the brain, cerebral neurasthenia, organic disease of the brain* (tumors, especially of the vermiform process) and *cord* (*multiple sclerosis* and *locomotar ataxia*), dis-

eases of the *stomach* (*vertigo a stomacho læsa*), diseases of the *ear* (*vertigo ab auræ læsa*), and from the action of certain toxic agents (*lead, alcohol, belladonna*).

Ménière's Disease (*labyrinthine vertigo*) is an affection depending upon disease of the internal ear, and characterized by *paroxysmal vertigo*, associated with *deafness* and ringing in the ears (*tinnitus aurium*).

Ringing or subjective noises in the ear may be classified as central and peripheral. To the former belong those noises produced exclusively by pathological changes of the structures of the labyrinth; and peripheral, to those due to causes external to the labyrinthal structures. Noises originating in the labyrinth may be caused by exudations, increased pressure, anæmic and hyperæmic conditions, and from the action of drugs. These noises always accompany a disturbance in hearing. Catarrh of the middle ear frequently gives rise to entotic or subjective noises. Sometimes the noises come from the vessels, particularly the carotid, which passes in close proximity to the ear. Other noises emanate from the jugular fossa, and others are produced by muscular contraction, particularly of the masseter.

Trance is a condition in which the patient shows no apparent signs of life. It may occur after attacks of hystero-epilepsy.

Ecstasy is a condition in which the mind is completely absorbed with some delusion.

Somnambulism is a condition in which the patient executes automatically various feats—such as writing, talking, walking, etc. It occurs in hysteria, and under the influence of hypnotic suggestion.

Catalepsy. Vide p. 172.

DIAGNOSIS OF INSANITY.

Hallucination is the perception of objects which have no real existence. The hallucination may be of taste, smell, vision, hearing and tact. If a person sees men where there are no men, he has an hallucination of vision.

Illusion is the misinterpretation of the character of an object, which has no real existence. If a person sees a chair and calls it a man, he has an illusion.

Delusion has no direct connection with sensation in any form. It results from disturbances in reasoning. A person who asserts he is the Almighty labors under a delusion. An *expansive delusion* or a *delusion of grandeur* is one which exalts its possessor. The grandiose ideas may embrace the possession of fabulous wealth, the undertaking of gigantic enterprises, etc.

Imperative Conceptions are ideas which the patient knows are absurd, but which nevertheless occur to him and dominate his thoughts, and often direct his actions. When he is unable to recognize the absurdity of his conception, it becomes a delusion.

Morbid Impulse is an irresistible desire to commit an act, which the patient knows to be wrong.

Moral Insanity is a congenital or acquired condition, of which depravity and absence of moral understanding constitute the chief manifestations.

Periodical Insanity (*Folie circulaire*) is characterized by alternating attacks of melancholia and mania, or mania and melancholia, returning at stated intervals.

The chief forms of insanity are *mania, melancholia, monomania, dementia, idiocy* and *imbecility.*

Mania is increased mental and physical activity. All sensations are exalted. The patients cry, laugh, sing and dance. They are in constant motion. Usually there is no elevation of temperature. *Mania transitoria* is a sthenic variety of mania, which lasts but a few hours or a day.

Melancholia is characterized by mental depression. According to the degree of depression there are three forms of melancholia: 1. *simple;* 2. *agitated;* 3. *attonita.* In all forms illusions and hallucinations are common. In *melancholia attonita* the patients are stupid, and remain in one position however uncomfortable. Nutritive and vascular disturbances attend this kind of melancholia.

Monomania signifies partial insanity on one subject, the mental powers in other respects being normal. The

delusions of monomania may include an endless variety of ideas. A monomaniac may, from the force of delusions or impellant ideas, commit theft (*kleptomania*) or arson (*pyromania*).

Imperative Conceptions occur in persons of a neuropathic constitution, in whom they may develop whenever the general nutrition of the body is lowered. The patients may fear contamination (*mysophobia*), and avoid touching articles previously used. Some even forego eating, believing the food to be unclean. Cases of perverted sexual instinct may be included under this caption.

Dementia may be divided into the following forms:

1. *Senile dementia* which results from senile disturbances of nutrition occurring in the brain. It is manifested by defective memory, apoplectic attacks, vertigo, insomnia, and mental depression. Ordinary insanity occurring in old people, must not be confounded with senile dementia.
2. *Terminal dementia* is weakness of the mind resulting from uncured cases of mental disease.
3. *Paralytic dementia* (general paralysis of the insane) is a chronic disease of the brain and its membranes, marked by psychical and motor disturbances, leading to dementia and paralysis. In the *prodromal stage* the following symptoms are usually present: irritability, loss of memory, egoism, apoplectiform attacks, tremor of the tongue and lips, slowness and hesitancy of speech, and inequality of the pupils.

Symptoms of second stage: mental excitement, insomnia, delusions of grandeur, intensification of prodromal symptoms, spastic or tabetic gait, and monoplegic or hemiplegic paralysis. Syphilis is the chief etiological factor aided by excesses of all kinds. The average duration of the disease is three or four years, and is invariably fatal, although lucid intervals or remissions may occur lasting several months.

Idiocy is a result of arrested brain development and is often associated with deformities and malformations of the body. The following forms of idiocy are recognized:

1. *Genetous idiocy* which results from intrauterine dis-

turbances. Two-thirds of the children die of consumption. In 517 cases examined by Howe, 21 were blind, 12 were deaf, 23 had some deformity of the mouth and nose, 54 had deformed hands or feet, and in 96 cases paralyses were present. They usually have the vaulted palate, protruding jaw and teeth, and retain an infantile appearance.

2. *Microcephalic idiots* have a narrow head tapering toward the top.

3. *Eclampsic idiocy* is produced by the convulsive seizures of infancy and childhood.

4. *Epileptic idiocy* is produced by epilepsy in infancy or childhood.

5. *Hydrocephalic idiocy* is produced by hydrocephalus.

6. *Paralytic idiocy* follows a celebral lesion.

7. *Traumatic idiocy* is produced by traumatism.

8. *Idiocy by deprivation* occurs when a child at birth or early life is deprived of two or more of its senses.

9. *Cretinism.*—Cretinoid idiots are confined to definite localities, usually in closed narrow mountain passes.

Cretins have a stupid, apathetic appearance, short, broad features, large mouth, hands and feet, thick lips and tongue (*macroglossia*), harsh voice, shuffling gait and *goitre* (characteristic). Cretinism differs from idiocy in its susceptibility to treatment.

Imbecility is a less profound arrest of the mental processes as compared with idiocy. Imbeciles are susceptible of more or less education.

RELATION OF VARIOUS DISEASES AND FUNCTIONS TO INSANITY.

Hypochondria.—In individuals predisposed to mental and nervous diseases, disturbances of the physical functions may lead to a condition of the mind in which the patient constantly fixes his attention on the morbid sensations. If he suffers from dyspepsia, he imagines he has cancer of the stomach. Such a condition is known as hypochondria. Hypochondria becomes *melancholia* when the imaginary troubles of the patient are actual delusions out of which he cannot be reasoned. Hypochondria is a much abused sub-

terfuge employed by the physician when he is incapable of detecting a cause for the disease. All hypochondriacs have a tangible reason for their complaints.

Epilepsy.—Insanity may follow the convulsions (*post-epileptic insanity*), or it may precede them. After an epileptic attack, the patient may be in a state of irritability and excitement (*post-epileptic delirium*); he may have maniacal attacks or hallucinations.

Hysteria.—In hysterical individuals, there is always a maniacal element in which the self-control, moral element or the feelings are involved. The insane manifestations may be mania, melancholia, or dementia. The diagnosis is based on the history of the case and the presence of *hysterical stigmata* (disturbances of sensation and motion, globus, etc.). *Hystero-Epilepsy* may simulate true epilepsy. The following are the chief points of differentiation:

Epilepsy: no apparent cause for attack, sudden onset, biting of tongue, of short duration, micturition and defecation may occur during the attack, convulsion is manifested by rigidity followed by jerking. Rise of temperature common.

Hystero-Epilepsy: caused by emotion, gradual onset, the convulsion consists of rigidity alone leading to arching of the back, the tongue is rarely bitten, the convulsions are of longer duration, micturition and defecation rarely occur, firm pressure over the ovaries may inhibit or excite an attack. There is only exceptionally a rise of temperature.

Chorea.—In an extreme form of this affection, which is more common in adult women and may develop during pregnancy (usually during the first five months), there may be maniacal excitement (*chorea insaniens*). Psychical disturbances are common in chorea and are manifested by irritability of temper, emotional outbreaks and mental enfeeblement.

Huntingdon's Chorea (chronic chorea) is an hereditary affection occurring in adult life, and is commonly followed by some mental disorder which terminates in dementia.

Physiological Crises (puberty, maternity, menopause, senility).

Puberty.—At this period of life in both sexes, a moral perversity may occur, manifested by a sudden disposition to steal, lie, swear, etc. It may be of long duration and finally result in incurable insanity. In girls, when menstruation is first established, excitement with delusions may occur.

Maternity.—About fifteen per cent. of cases of insanity among women occur during the performance of the functions of pregnancy, parturition, and lactation.

Puerperal mania usually occurs two or three weeks after delivery. The attack is preceded by excitement, insomnia, constriction of the head and disturbance of vision. The insanity, when fully developed, may amount to delirious mania.

Menopause.—Nervous and mental symptoms of a mild character are common to all women during this period. Mental disorder may occur during any stage of the menopause, and is characterized by the various shades of melancholia.

Senility.—In men between the ages of 55 and 65, there is a period corresponding to the menopause of women. In this period retrograde changes involving the arteries and cortex of the brain occur, which conduce to mental disturbances.

Alcoholism.—The long-continued use of alcohol may lead to all grades of mental impairment from enfeeblement of the mind to dementia (*alcoholic dementia*).

Dipsomania constitutes a manifestation of chronic alcoholism in which the patient is impelled periodically to indulge in a morbid craving for alcohol.

Delirium Tremens (*mania a potu*) follows the long-continued use of alcohol. It may occur during the time the patient is indulging himself in alcohol or after its sudden withdrawal. Sometimes an accident or acute infectious disease (*pneumonia*) may determine the onset of the delirium. *Symptoms:* The patient talks constantly and has hallucinations of sight and hearing. He sees rats, mice, and hears unusual sounds, there is muscular tremor, rise of temperature and insomnia. Abatement of restlessness and tendency to sleep usually terminate the attack. Recovery is usual.

The effects of alcohol as a tissue poison is manifested by degenerative changes of the parenchyma of the viscera, thickening and fibroid changes of the arteries, and fatty changes due to diminished oxidation of the tissues, since alcohol is consumed in lieu of fat. The habitual use of alcohol leads specially to the following affections: hemorrhagic pachymeningitis, alcoholic neuritis, paralytic dementia, catarrh of the stomach, cirrhosis of the liver, arterio-sclerosis and granular kidney.

Toxic Insanities result from the action of certain toxic agents, such as *morphine*, *lead*, and *mercury*.

Organic Dementia has its anatomical substratum in cerebral organic affections, such as embolism, thrombosis, softening and apoplexy. The mental disturbances result from defective nutrition of the cortical centers. The mental alienation is usually characterized by loss of memory, although mental exaltation or depression may be present. The patients are usually stupid and silly.

Syphilis may relead to insanity as the result of the luetic poison on the brain (*cerebral syphilis*). Cerebral syphilis may first show itself by sudden and violent delirium. Some cases show a prolonged torpor. Syphilis not only predisposes to, but directly produces *dementia paralytica*. *Syphilophobia* is a condition in which the patient imagines he has acquired syphilis, or believes that every simple skin lesion may be a manifestation of it.

Etiology of Insanity.—The causes may be *predisposing* and *exciting*. To the former belong the excessive use of alcohol, tobacco, coffee and anxiety. Insanity is equally frequent in both sexes. Before puberty insanity is rare; if occurring before this period it may be congenital or acquired from certain habits, as masturbation, or from diseases, such as epilepsy, chorea, infectious diseases, etc. The greater percentage of insanities occurs, in women, between 20 and 35; and in men, between 30 and 50 years of age. Heredity is an important factor in the etiology of insanity.

Exciting causes.—Fright, worry, organic affections of the brain and the acute infectious diseases.

Prognosis of Insanity.—In simple cases of *mania*, the prognosis is favorable. If recovery does not occur, it is

followed by mental weakness. A large number of cases of *melancholia* recover, but the prognosis is not so favorable as in mania. Recovery is slow and marked by periods of exacerbation. If recovery does not take place, it terminates into either chronic melancholia or dementia. *Senile dementia* lasts from about one to three years. The prognosis of *hypochondria* is always doubtful; it begins and progresses slowly and may have remissions. *Dementia paralytica* is unfavorable; patients rarely living over two, three or four years.

Topography of the Brain.—To locate the fissures and convolutions of importance on the surface of the brain, the following method of Reid is recommended for its simplicity. Reid's base line runs backward from the infra-orbital ridge through the middle of the external auditory meatus, and prolonged to the middle line of the head posteriorly. The *fissure of Sylvius* runs from a point an inch and a quarter behind the external angular process of the frontal bone to a point three-fourths of an inch below the most prominent point of the parietal eminence. Measuring from above backward, the first three-fourths of an inch will represent the main fissure; the rest indicates the horizontal limb. The ascending limb starts at a point three-fourths of an inch back of the anterior extremity; that is, two inches behind and slightly above the external angular process, and runs vertically upward and forward about one-fourth of an inch.

To find the *fissure of Rolando*, draw the base line and the lines for the great longitudinal fissure (an imaginary line corresponding to the course of this fissure) and the fissure of Sylvius. Then draw two lines perpendicular to the base line; one from the depression in front of the external meatus and the other from the posterior border of the mastoid process at its root. We now have on the surface of the head a four-sided figure, bounded above and below by the lines for the longitudinal fissure and the horizontal limb of the fissure of Sylvius, respectively, and in front and behind by the two perpendicular lines just described. Now draw a diagonal line from the posterior superior angle to the anterior inferior angle. This corresponds to the *fissure of Rolando*, which, as a rule, does not quite join the fissure of Sylvius.

LOCALIZATION OF THE FUNCTIONS OF THE SEGMENTS OF THE SPINAL CORD.—(*Starr.*)

SEGMENT.	MUSCLES.	REFLEX.	SENSATION.
2d and 3d cervical	Sterno-mastoid, trapezius, scaleni and neck, diaphragm.	Hypochondrium (?); sudden inspiration produced by sudden pressure beneath the lower border of ribs.	Back of head to vertex; neck.
4th cervical	Diaphragm, deltoid, biceps, coraco-brachialis, supinator longus, rhomboid, supra- and infraspinatus.	Pupil, 4th to 7th cervical; dilatation of the pupil produced by irritation of the neck.	Neck, upper shoulder, outer arm.
5th cervical	Deltoid, biceps, coraco-brachialis, brachialis anticus, supinator longus, supinator brevis, rhomboid, teres minor, pectoralis, serratus magnus.	Scapular, 5th cervical to 1st dorsal; irritation of the skin over scapula produces contraction of the scapula muscles, supinator longus; tapping its tendon in wrist produces flexion of forearm.	Back of shoulder and arm; outer side of arm and forearm, front and back.
6th cervical	Biceps, brachialis anticus, pectoralis (clavicular part), serratus magnus, triceps, extensors of wrist and fingers, pronators.	Triceps, 5th to 6th cervical; tapping elbow tendon produces extension of forearm; posterior wrist, 6th to 8th cervical; tapping tendon causes extension of hand.	Outer side of forearm, front and back; outer half of hand.
7th cervical	Triceps (long head), extensors of wrist and fingers, pronators of wrist, flexors of wrist, subscapular, pectoralis (costal part), latissimus dorsi, teres major.	Anterior wrist, 7th to 8th cervical; tapping anterior tendons causes flexion of wrist; palmar, 7th cervical to first dorsal; striking palm causes closure of fingers.	Inner side of back of arm and forearm; radial half of hand.
8th cervical	Flexors of wrist and fingers, intrinsic muscles of hand.	— —	Forearm and hand, inner half.
1st dorsal	Extensors of thumb, intrinsic hand muscles, thenar and hypothenar eminences.	— —	Forearm, inner half; ulnar distribution to hand.

SEGMENT.	MUSCLES.	REFLEX.	SENSATION.
2d to 12th dorsal	Muscles of back and abdomen, erectores spinæ.	Epigastric, 4th to 7th dorsal; tickling mammary region causes retraction of the epigastrium; abdominal, 7th to 11th dorsal; striking side of abdomen causes retraction of belly.	Skin of chest and abdomen in bands running around and downwards corresponding to spinal nerves; upper gluteal region.

SPINAL LOCALIZATION.

1st lumbar	Ilio-psoas, sartorius, muscles of abdomen.	Cremasteric, 1st to 3d lumbar; striking inner thigh causes retraction of scrotum.	Skin over groin and front of scrotum.
2d lumbar	Ilio-psoas, sartorius, flexors of knee (Remak) quadriceps, femoris.	Patella tendon; striking tendon causes extension of leg.	Outer side of thigh.
3d lumbar	Quadriceps femoris, inner rotators of thigh, abductors of thigh.	—— ——	Front and inner side of thigh.
4th lumbar	Abductors of thigh, adductors of thigh, flexors of knee (Ferrier), tibialis anticus.	Gluteal, 4th and 5th lumbar; striking buttock causes dimpling in fold of buttock.	Inner side of thigh and leg to ankle; inner side of foot.
5th lumbar	Outward rotators of thigh, flexors of knee (Ferrier), flexors of ankle, extensors of toes.	—— ——	Back of thigh, back of leg, and outer part of foot.
1st and 2d sacral	Flexors of ankle, long flexors of toes, peronei, intrinsic muscles of foot.	Plantar; tickling sole of foot causes flexion of toes and retraction of leg.	Back of thigh, leg, and foot, outer side.
3d to 5th sacral	Perineal muscles.	Foot reflex, Achilles tendon; over extension of foot causes rapid flexion, ankle clonus, bladder and rectal centres.	Skin over scrotum, anus, perineum, genitals.

DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM.

DISEASES OF THE NERVES.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
1. NEURITIS.		
Inflammation localized in a single nerve. The sheath may be involved or the axis cylinders (*parenchymatous neuritis*).	Slight constitutional disturbances, pain along the nerve, muscular power is impaired and trophic disturbances are present.	Pain is constant. There is first hyperæsthesia and later anæsthesia of the affected parts. The affected nerve is tender and thickened.
2. MULTIPLE NEURITIS, (Polyneuritis.)		
Inflammation involving a large number of nerves.	The onset is like that of an acute infectious disease. Pain, numbness, loss of power and the reflexes, tenderness of the nerve trunks and degenerative atrophy of the muscles.	Like the foregoing, besides the constitutional symptoms. *Wrist drop*, when the upper, and *foot drop*, when the lower extremities are involved. *Steppage gait;* the foot is raised high, toe raised, and the heel brought down first.

DISEASES OF THE SPINAL CORD AND MENINGES.

1. PACHYMENINGITIS CERVICALIS.		
Inflammation of the *Dura mater* caused by caries or tuberculosis of the vertebræ, syphilis, etc.	The first stage is marked by pains, the second stage by paralysis. The disease usually lasts over years. Recovery rare.	Pains and stiffness in the neck, paræsthesias in the arms followed by paralysis.
2. LEPTOMENINGITIS.		
Inflammation of the soft spinal meninges, which may be acute or chronic.	Moderate fever, pains in the back, rigidity of spinal muscles and paralysis.	Stiffness of the muscles of the back and tenderness of the vertebræ.
3. HEMATORRHACHIS. (Meningeal Apoplexy.)		
The hemorrhage may be *extra-meningeal* (between the dura and spinal canal) or *intra-meningeal* (within the dura).	The symptoms resemble spinal meningitis, only that the onset is very sudden. There is complete freedom from disturbances of consciousness.	If hemorrhage is in *lumbar cord*, the legs, bladder and rectum suffer. In *dorsal cord*, irritation of intercostal nerves. In *cervical cord*, motor and sensory disturbances in upper extremities.

DISEASES OF THE SPINAL CORD—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
4. CAISSON DISEASE. (Diver's Paralysis).		
Liberation in the spinal cord of bubbles of nitrogen which have been absorbed by the blood under high pressure, resulting in myelitis and hemorrhages.	Found in divers and workers in caissons. Paraplegia which follows on returning from the compressed atmosphere to the surface.	Sudden onset of the symptoms.
5. ACUTE MYELITIS.		
When a transverse section of the cord is involved it is called *transverse myelitis;* when the gray matter is involved, *central myelitis:* when a large vertical section of the cord is involved, *diffuse myelitis.*	*Acute central myelitis:* sensory and trophic disturbances, paralysis of bladder and rectum, rapid wasting, electrical changes and fever. *Transverse myelitis;* fever, girdle pain at level of disease, flabby paralysis of the muscles, bed sores, and anæsthesia.	*Transverse myelitis of the cervical region;* paralysis of motion and sensation in upper extremities, vomiting, slow pulse, contraction of the pupils. *Myelitis of the dorsal region;* motor and sensory paralysis in the lower extremities.
6. POLIO-MYELITIS ANTERIOR ACUTA.		
The anterior horns of the gray matter are atrophied, the motor cells are degenerated. The anterior nerve roots and affected muscles are atrophied. The cervical or lumbar enlargements are usually affected.	Usually occurs during the first three years of life. Onset is abrupt. No sensory disturbances. The paralysis is confined to certain groups of muscles in the upper and lower extremities.	Acute disease, occurring in young children with fever, loss of power in certain muscles and rapid atrophy. The reflexes are absent.
7. LANDRY'S PARALYSIS. (Acute Ascending.)		
Occurs in males between the twentieth and thirtieth years. The disease is like a peripheral neuritis, although no definite demonstrable lesions have been found.	An advancing paralysis beginning in the legs and extending rapidly to the trunk and arms, ending usually in implication of the respiratory muscles.	Rapidly advancing motor paralysis without affecting the sphincters, without wasting or electrical changes in the muscles, absence of trophic lesions and fever.
8. SPASTIC PARAPLEGIA. (Lateral Sclerosis.)		
In *primary spastic paraplegia* there is primary sclerosis of the lateral tracts. *Secondary spastic paraplegia* is secondary sclerosis of the lateral tracts after any transverse lesion of the cord.	The patient complains of a tired feeling and stiffness in the legs. The reflexes are so increased that the slightest touch upon the patella tendon results in an active knee jerk.	Characteristic gait (p. 3). Loss of power with spastic contraction of the muscles of the lower extremities, exaggerated knee jerk, presence of ankle clonus. No muscular wasting or disturbance of sensation.

DISEASES OF THE SPINAL CORD—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
9. HEREDITARY ATAXIA. (Friedreich's Ataxia.)		
Extensive sclerosis of the posterior and lateral columns of the cord.	A family disease occurring in early life and characterized by almost the same clinical feature as tabes dorsalis.	A disease of childhood marked by incoördination, nystagmus, scoliosis, scanning speech and talipes equinus.
10. LOCOMOTOR ATAXIA. (Tabes Dorsalis.)		
The peripheral nerves and posterior roots of the cord are degenerated. The posterior columns of the cord are sclerotic, especially in the dorsal and lumbar regions. The membranes over the posterior columns are inflamed.	*Pre-ataxic stage.* Lightning pains in the legs, ptosis, Argyll-Robertson pupil, loss of the knee jerk. *Ataxic stage.* Symptom of Romberg (p. 170), ataxic gait (p. 3), lightning pains continue, sensory disturbances and gastric crises. *Paralytic stage.* Loss of motor power.	Lightning pains, absence of knee jerk, Argyll-Robertson pupil, marked incoördination with no loss of muscular power, diplopia, optic atrophy, impotency and Romberg's symptom.
11. SYRINGO-MYELIA.		
There is an overgrowth of embryonal neuroglia in which degeneration occurs, leading to a cavernous condition of the cord. It usually involves the posterior portion of the cord, and extends into one posterior cornu.	A disease of slow development, persisting a long time, appearing about the time of puberty, and characterized by neuralgic pains, wasting of the muscles, and loss of painful and thermic sensations.	Paralysis of an amyotrophic type, with retention of tactile and loss of thermic and painful sensations.
12. COMPRESSION MYELITIS.		
Interference of the functions of the spinal cord by slow compression, and produced by any of the following causes: caries of the spine, tumors, trauma, and aneurism.	The developed symptoms may be due to compression of the vertebral column (deformity), nerve roots (pains), or cord (paralysis).	Tenderness of the vertebral column, painful areas of the skin which are anæsthetic (*anæsthesia dolorosa*) and deformity of the spine. The symptoms develop slowly.
13. BROWN SEQUARD'S PARALYSIS.		
Destruction of one-half the cord by fracture, caries, tumors, stab-wounds, etc.	Spinal hemiplegia. Loss of sensation on the side opposite to the lesion. The tactile sensation is usually unimpaired. The reflexes are increased on the side of the lesion.	Motor paralysis on the side of the lesion and a sensory paralysis on the opposite side. There is often hyperæsthesia on the side of the motor paralysis.

DISEASES OF THE SPINAL CORD—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
14. AMYOTROPHIC LATERAL SCLEROSIS.		
Simultaneous implication of the ganglionic cells of the gray anterior horns and the pyramidal or lateral tracts of the cord.	The disease is first noticed in the arm, and is attended by paresis and wasting of the muscles of the hand and fingers. Other muscles become atrophied. At a later period of the disease the patient has bulbar symptoms.	The co-existence of muscular atrophy, increase of the tendon reflexes, bulbar symptoms and absence of sensory and bladder disturbances.
15. POLIOMYELITIS ANTERIOR CHRONICA. (Progressive Muscular Atrophy.)		
Atrophy of the muscles, atrophy of the anterior roots, and atrophy of the ganglion cells of the anterior horns. The essential lesion is of the ganglion cells in the anterior horns.	It is a disease of adults occurring after the thirtieth year, and manifested anatomically by degeneration of the ganglion cells, and clinically by loss of power and atrophy of the corresponding muscles. The muscles of the hand are first affected.	Muscular atrophy, in the adult beginning in the thumb muscles and slowly involving the interossei and lumbricales.
16. GLOSSO-LABIO-LARYNGEAL PARALYSIS. (Bulbar Paralysis.) (Duchenne's Disease.)		
Degenerative atrophy of the nuclei of nerves that take their origin on the floor of the fourth ventricle. The degeneration is rarely primary but secondary to some disease of the motor path.	Impairment of speech, paralysis and atrophy of tongue, lips, and pharynx, inability to swallow, dribbling of saliva, suppression of the voice, difficult respiration and disturbed cardiac rhythm.	Disturbances confined to the motor functions presiding over the muscles of the tongue, lips, pharynx and larynx. There are no sensory disturbances.

DISEASES OF THE BRAIN AND MENINGES.

1. HEMORRHAGIC PACHYMENINGITIS.		
Found in insanity and chronic alcoholism. Subdural hemorrhage in connection with atrophy of the convolutions.	Headache is a prominent symptom, although the affection may exist without symptoms.	Headache, abuse of alcohol and disturbances of intellection.

DISEASES OF THE BRAIN—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
2. SIMPLE LEPTO-MENINGITIS.		
The convexity of the pia is usually affected. It presents all the evidences of inflammation. It may be caused by traumatism, rheumatism, and other infectious diseases, and extension of inflammation from ear.	Moderate fever, retraction of the head, convulsions, headache and coma. The prognosis is usually unfavorable.	Headache, stiffness and retraction of the neck muscles and vomiting.
3. TUBERCULOUS MENINGITIS.		
The meninges at the base of the brain are most involved (*basilar meningitis*). The inflammation of the membrane is a tuberculous one.	Disease of childhood usually. Begins insidiously with nervous symptoms followed by headache, shrill scream (*hydrocephalic* cry), rigidity of neck muscles, fever, delirium, convulsions and coma.	Acute beginning with epileptiform convulsions, trismus, strabismus, rigidity of the muscles of the neck, vomiting, neuritis and tubercles in the choroid.
4. CEREBRAL ANÆMIA.		
Deficient supply of blood to the brain may be associated with general anæmia, aortic stenosis and degeneration of the cerebral blood-vessels.	Pallor of the face, vertigo, dimness of vision, dilatation of the pupil, syncope, failure of memory, etc. The symptoms are ameliorated after inhalation of nitrite of amyl.	The ophthalmoscope shows retinal anæmia. Head symptoms associated with anemia and improving after removal of the latter.
5. CEREBRAL HYPERÆMIA.		
Congestion of the brain may be *active*, as occurs in fevers, or *passive* from obstruction in the cerebral sinuses and veins.	There are no definite symptoms, in fact the clinical picture may resemble that of cerebral anæmia.	The ophthalmoscope shows retinal hyperæmia, There is usually headache and congestion of the face.
6. CEREBRAL HEMORRHAGE. (Apoplexy.)		
The blood-vessels are atheromatous and occupied by miliary aneurisms. The most frequent seat of the hemorrhage is in the internal capsule. The hemorrhage may be *cerebral*, *meningeal*, or *intraventricular*.	Patient, usually without warning, becomes suddenly unconscious, with flushed face and stertorous breathing, rotation of the eyes to one side (*conjugate deviation*) and paralysis. Sensation is unaffected unless posterior limb of the internal capsule is involved.	Met with in aged individuals in whom the arteries are atheromatous. Sudden loss of consciousness accompanied with paralysis.

DISEASES OF THE BRAIN—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
7. CEREBRAL EMBOLISM AND THROMBOSIS. (Cerebral Softening.)		
Emboli are most frequent in a branch of the left middle cerebral artery. The majority of emboli are derived from valves of the heart in endocarditis. Thrombi are most common in the middle cerebral and basilar arteries.	In embolism the onset is sudden without prodromes. Embolism of the left middle cerebral artery causes hemiplegia with aphasia. In thrombosis the symptoms (headache, vertigo, insomnia, etc.) are developed slowly.	*Diagnosis of embolism:* presence of a cardiac lesion, occurrence in the young, absence of prodromes and no unconsciousness during the attack.
8. THROMBOSIS OF THE CEREBRAL VEINS AND SINUSES.		
Primary thrombosis is rare. *Secondary thrombosis* follows extension of inflammation from neighboring parts, such as diseases of the ear, fracture and erysipelas.	The onset of the disease is usually sudden and marked by symptoms of septicemia. There is fever, rigors, pain in the occipital region and the neck.	The diagnosis is difficult, and can only be made if, in conjunction with the general symptoms, signs are added peculiar to sinus thrombosis as, for instance, œdema of the eyelids and congestion of the retina point to involvement of the cavernous sinus.
9. CEREBRAL PARALYSIS IN CHILDREN.		
Atrophy and sclerosis of the cerebral convolutions and *porencephalus* (a cystic condition of the cortex). Difficult labor, injury with the forceps and infectious diseases are etiological factors.	The paralysis may be hemiplegic, diplegic, or paraplegic. It occurs at birth or during the first few years of life. The children may be imbeciles or idiots.	In more than half the cases, the paralysis is preceded by partial or general convulsions and loss of consciousness. Fever is usually present.
10. TUMORS OF THE BRAIN.		
The most common varieties are tubercle, gumma, glioma, cysts, sarcoma and carcinoma.	Headache with localized tenderness, vomiting, optic neuritis, vertigo, failure of memory and symptoms resulting from pressure (paralysis, convulsions, etc.).	Headache, optic neuritis and vomiting. The character and location of the tumor must be determined along with the existence of the growth.
11. ABSCESS OF THE BRAIN.		
In four-fifths of all cases, the abscess is solitary. The temporosphenoidal lobe and the cerebellum are the most frequent seats.	A history of traumatism followed by fever, headache, delirium, vomiting and rigors. There may be otorrhea or some suppurating disease.	History of traumatism or existence of some suppurating disease followed by all the symptoms of a brain tumor plus the fever.

DISEASES OF THE BRAIN—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
12. INSULAR SCLEROSIS.		
Sclerosed patches diffusely distributed in the brain and cord, and consisting of an increased growth of connective tissue which destroys the medulla of the nerves, the axis cylinders persisting.	A disease occurring in middle-aged persons, of chronic course, and characterized by volitional tremor, scanning speech, nystagmus and vertigo.	Tremor on attempting movements, nystagmus, scanning speech and spastic weakness of the legs.
13. DEMENTIA PARALYTICA. (General Paresis.)		
Cerebral membranes are thickened and adherent to the brain, the cortex is atrophied and there is an increase of connective tissue and degeneration of nerve fibres and ganglionic cells.	A chronic, progressive meningo-encephalitis associated with psychical and motor disturbances leading to paralysis and dementia.	Delusions of grandeur, trembling of the tongue, slow speech, unequal pupils, shuffling gait and Argyll-Robertson pupil.
14. HYDROCEPHALUS.		
Accumulation of fluid within the ventricles of the brain, associated with a large head, sutures and fontanelles. The condition may be congenital or acquired.	Congenital hydrocephalus occurs in the first few years of life associated with impaired intelligence, increased reflexes, convulsions and various motor phenomena.	Globular enlargement of the head, open sutures and fontanelles and impaired intelligence. The congenital cases usually die within the first four or five years.

FUNCTIONAL NERVOUS DISEASES.

1. ACUTE DELIRIUM. (Bell's Mania.)		
There are no constant pathological lesions. Acute infection is a possible factor in the etiology.	A disease running a rapidly fatal course, associated with fever, delirium and insomnia.	Abrupt onset, delirium, fever and fatal termination within three weeks.
2. PARALYSIS AGITANS. (Parkinson's Disease.)		
No constant lesions, although the anatomical substratum of its occurrence is due to the usual lesions of cerebro-spinal senility.	A chronic affection, more frequent in men than women, and rarely occurring before the fortieth year. It is characterized by muscular weakness, tremors and rigidity.	Gait (p. 3), tremor when the muscles are at repose, weakness and rigidity of the muscles, expressionless face and shrill and piping voice.

FUNCTIONAL NERVOUS DISEASES—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
3. ACUTE CHOREA.		
No constant lesions. Emboli in the cerebral vessels. Endocarditis is believed by some to be the cause.	A disease of children, manifested by irregular involuntary contractions of the muscles. It is more frequent in females.	Occurrence in young patients of female sex; contractions of muscles disappear during sleep; contractions occur independent of the will.
4. INFANTILE CONVULSIONS. (Eclampsia.)		
May occur from debility, rickets, peripheral irritation, as dentition, fever and cerebral congestion.	The convulsive seizure resembles that of epilepsy, but differs from the latter, that when the cause is removed the convulsions do not recur.	Convulsions associated with a loaded stomach or as a prelude to an infectious disease, in which instance it is accompanied by fever and vomiting.
5. EPILEPSY.		
No definite lesion. The following causes may be cited: heredity, chronic alcoholism in the parents, syphilis and trauma.	Attacks of unconsciousness with or without convulsions. The former is known as *grand mal*, the latter as *petit mal*. The attacks are preceded by a sensation known as *aura*.	Abrupt loss of consciousness followed by tonic and clonic spasms ending in coma.
6. HEMICRANIA. (Migraine.)		
The nature of the disease is not known. It is related to epilepsy, and consists of a nerve storm or periodic discharge from sensory centers. The early symptoms are supposed to be due to vaso-constrictor and the later symptoms to vaso-dilator influences.	A paroxysmal affection manifested by unilateral headache and associated with visual and gastric disturbances. Women and neurotic members of a family are predisposed. Nausea and vomiting are frequent symptoms.	Periodical unilateral headache, the patient remaining well during the intervals of the attacks. *Spastic hemicrania* is associated with pallor, and *paralytic hemicrania* with redness of the face.
7. TETANY.		
No lesion. Tetany may be: 1, *epidemic;* 2, may follow *lactation* or *chronic diarrhea;* 3, after removal of the thyroid gland; 4, it may be associated with dilatation of the stomach.	It is manifested by tonic spasms of the extremities. The irritability of the nerves is increased, and pressure on a nerve trunk or artery will cause a spasm.	See pages 171 and 180.

FUNCTIONAL NERVOUS DISEASES—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
8. HYSTERIA.		
There are no anatomical changes. It is a state in which ideas control the body and produce morbid changes in its functions. It is an increased excitability of the whole nervous system.	It is a functional disease with motor (paralysis, spasms, convulsions), sensory (anæsthesia and hyperæsthesia, etc.), and psychical symptoms.	The symptoms develop and disappear abruptly, are unaccompanied by general ill-health, are intensified or inhibited by emotional excitement.
9. TRAUMATIC NEUROSIS. (Railway Spine.)		
The condition following a shock may present the symptoms of neurasthenia or hysteria, or both, or may be the result of organic disease of the brain and cord.	*Symptoms of neurasthenia:* headache, tired feeling, irritability, numbness in the extremities, disturbed digestion. *Symptoms of hysteria:* sensory disturbances, tremor and limitation of the field of vision.	Organic changes are indicated by optic atrophy, bladder symptoms, tremor and exaggerated reflexes. The history of a shock preceding the symptoms.
10. NEURASTHENIA.		
No demonstrable anatomical lesions. It is a weakness or exhaustion of the nervous system.	According to the clinical picture: *Cerebral neurasthenia:* depression of spirits, vertigo, insomnia, irritability and hysterical manifestations. *Spinal neurasthenia:* pain in the back, weakness on exertion, and local tenderness of the spine.	Absence of symptoms pointing to organic mischief. The diagnosis of neurasthenia should only be made after an exclusion by systematic examination of organic disease. Neurasthenia is hysteria occurring in man.
11. JACKSONIAN EPILEPSY.		
Irritative lesions in the motor zone of the cerebral cortex, such as tumor, softening, meningitis, abscess and hemorrhage.	Spasms occurring in definite muscle groups, as the face, arm or leg with retention of consciousness; as a prelude to the spasms there may be numbness in the part.	Paroxysmal spasms confined to definite muscle groups with perfect consciousness.
12. ASTASIA ABASIA.		
A neurosis related to hysteria.	The patient is unable to stand erect or walk properly, notwithstanding muscular strength, coördination and sensation are intact.	When the patient stands, the legs give way as if made of cotton.

FUNCTIONAL NERVOUS DISEASES—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
13. TIC CONVULSIF. (Tourette's Disease.)		
A neurosis related to hysteria and occurring usually in young children with a neurotic family history.	Involuntary muscular movements of the face, arm or other groups of muscles, accompanied by explosive utterances in which bad language is often used (*coprolalia*).	Involuntary muscular movements, explosive utterances and mental disturbances.
14. ATHETOSIS.		
It is usually symptomatic of a cerebral or spinal affection, or it may occur idiopathically.	Rhythmical movements of the fingers and toes which cannot be controlled by the patient, and persist without interruption during waking and sleeping.	Athetosis is usually a symptomatic affection. The athetoid movements continue during sleep and are usually uninfluenced by treatment.

SYNOPTIC TABLE OF PARALYSES.

	SPINAL PARALYSIS.	PERIPHERAL PARALYSIS.	CEREBRAL PARALYSIS.	MYOPATHIC PARALYSIS.	FUNCTIONAL PARALYSIS.
ETIOLOGY.	Myelitis, tumors of the cord, compression of the cord, hemorrhage and traumatism.	Trauma, neuritis, neuroma, chemical and thermic irritants and infectious diseases.	Thrombosis, embolism, abscess, tumors, and hemorrhage.	Atrophy of the muscles independent of the nervous system. The affection is hereditary, and sets in usually before puberty.	Hysteria.
ELECTRO-DIAGNOSIS.	Reaction of degeneration is present in all paralyses produced by disease of the ganglionic cells, in the anterior horns of the gray matter.	Reaction of degeneration.	Electrical irritability unchanged, unless the cerebral nuclei are implicated.	There may be diminished electrical irritability, but the reaction of degeneration is never present.	There is no reaction of degeneration.
DISTRIBUTION OF PARALYSIS.	Hemiplegia, paraplegia, hemi-paraplegia and paralysis of special nerve roots. Paraplegia is the usual form.	Limited to the muscles supplied by the affected nerves.	In cortical lesions monoplegia may occur; as a rule there is hemiplegia. Facial paralysis, if present, is only partial, involving the lower portion of the nerve.	Muscles of the upper arm and shoulder, gluteal, thigh and facial muscles.	Hemiplegia is usually on the left side; the face is not affected, but the leg suffers most. *Paraplegia* is more common than the former. The loss of power is not absolute.

SYNOPTIC TABLE OF PARALYSES.—*Continued.*

	SPINAL PARALYSIS.	PERIPHERAL PARALYSIS.	CEREBRAL PARALYSIS.	MYOPATHIC PARALYSIS.	FUNCTIONAL PARALYSIS.
CONDITION OF THE MUSCLES.	Atrophy of the muscles, when the cells of the anterior horn are affected, or when the anterior nerve roots are separated from their trophic center.	Atrophy of the involved muscles.	No degenerative muscular atrophy, notwithstanding long continuance of the paralysis.	Atrophy in conjunction with hypertrophy of the muscles. Fibrillary contractions are absent.	No atrophy of the muscles.
DISTURBANCES OF SENSATION.	Disturbances of sensation are common, especially if the posterior nerve roots are implicated.	In a mixed nerve, *anæsthesia* limited to the distribution of the nerve.	Hemianæsthesia may be present, especially if the lesion is in the internal capsule. There may be no sensory disturbances.	There is usually no disturbance of sensation.	*Anæsthesia* is common and usually confined to one half the body. It is as a rule limited to the middle line and involves the deep parts.
REFLEXES.	In implication of the lateral column, the reflexes are increased, otherwise they are usually diminished or absent.	Absent, if the reflex circuit is destroyed by the nerve lesion.	The deep reflexes are increased on the paralyzed side, and ankle clonus may be present. The sphincters are not affected.	The tendon reflexes are present, but in cases where the muscle atrophy is pronounced they may be reduced.	The reflexes may be increased or normal.
CONCURRENT SYMPTOMS.	Girdle sensations, impairment of the bladder or rectum.	Absence of cerebral and spinal symptoms.	Psychical disturbances, headache, disturbances of vision and hearing, ophthalmoscopic changes, vertigo and aphasia.	The paralysis is always proportionate to the atrophy.	Hysterical symptoms. *Hysterical paraplegia* is not attended by implication of the bladder.

VASO-MOTOR AND TROPHIC DISEASES.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
1. RAYNAUD'S DISEASE. (Symmetrical Gangrene.)		
A vascular affection characterized by various grades of intensity, viz.: 1. Local syncope; 2. Local asphyxia; 3. Local gangrene. The first is produced by contraction of the blood-vessels, and the second by dilatation of the capillaries and small veins.	*Syncope.* The fingers or toes look like dead. *Asphyxia.* The toes or fingers are livid, swollen and painful. The disease is usually confined to the extremities.	The affection is usually symmetrical, and hemoglobinuria is not uncommon during an attack.
2. ANGIO-NEUROTIC ŒDEMA.		
A neurosis with an hereditary tendency, and caused by vaso-motor dilatation of the blood-vessels with transudation of serum.	Œdematous swelling appearing in various parts of the body, particularly in the face and hands.	Œdema is circumscribed and appears suddenly attended by gastro-intestinal distress.
3. ACROMEGALIA. (Marie's Disease.)		
Hypertrophy of the bones and pituitary body, and persistence of the thymus gland.	A dystrophy characterized by increased growth of the bones of the face and extremities and overlying tissues.	Egg-shaped face with the large end downward, dullness over the manubrium sterni due to persistence or enlargement of the thymus gland.
4. FACIAL HEMIATROPHY.		
Interstitial neuritis of the trigeminus nerve. The nature of the disease is doubtful.	Progressive wasting of the bones and soft tissues of one side of the face. It usually develops in childhood.	The affected side is sunken, and the skin shows a whitish-brown discoloration.
5. EXOPHTHALMIC GOITRE.		
Nothing definite. Enlargement and thickening of the cervical sympathetic ganglia have been observed.	A disease of early adult life, frequent in females and characterized by palpitation of the heart, protrusion of the eye-balls, and enlargement of the thyroid gland.	See page 181. One of three important symptoms may be absent. Tremor may be present and shows a great regularity of rhythm.

VASO-MOTOR AND TROPHIC DISEASES—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
6. MYXŒDEMA.		
Mucoid degeneration of the subcutaneous tissues, and atrophy of the thyroid gland.	A constitutional disease, more frequent in women than in men, and marked by swelling in the face and upper extremities, mental failure and dementia.	The swelling does not pit on pressure. Features are coarse, and over the cheeks there is a reddish patch. Temperature is subnormal, and there is atrophy of the thyroid gland.
7. SUNSTROKE. (Thermic Fever.)		
Early rigor mortis, congestion of the brain and lungs, and parenchymatous changes in the liver and kidneys.	After exposure to high temperatures, exhaustion, vertigo, nausea and headache followed by coma, hyperpyretic temperature with full and rapid pulse.	The mode of onset and high temperature enables the coma to be differentiated from apoplexy, alcoholism, etc.

DISEASES OF THE MUSCLES.

1. IDIOPATHIC MUSCULAR ATROPHY.		
Muscular wasting with or without initial hypertrophy, and occurring independently of the nervous system.	An hereditary disease of progressive character, presenting two chief types : 1, primary hypertrophy ; and 2, primary atrophy.	The primary myopathies involve the muscles of the calves, trunk, face or shoulder girdle. They occur usually before puberty. (See pseudo-hypertrophic paralysis).
2. MYOTONIA CONGENITA. (Thomsen's Disease.)		
The nature of the affection is unknown. The muscular fibres of the voluntary muscles are increased in width.	An hereditary disease characterized by tonic cramp or spasm of the muscles on attempted movement. The affection first appears in childhood.	Muscular spasms on attempting voluntary movements. The sensation and reflexes are normal. *Myotonic reaction* (on galvanic stimulation of the muscles, the contractures are slow and prolonged, lasting as long as thirty seconds).

DISEASES OF THE MUSCLES—Continued.

Morbid Anatomy.	*General Symptoms.*	*Characteristic Symptoms.*
3. PARAMYOCLONUS MULTIPLEX.		
The nature of the disease is unknown, although it may be regarded as an emotional neurosis of hysterical origin.	Clonic muscular spasms in the muscles of the face and extremities, usually symmetrical and appearing in paroxysms. The strength of the muscles and electrical excitability remain unaltered.	Clonic contractions of the muscles unaccompanied by sensory or motor disturbances.
4. GRAPHOSPASM. (Writers' Cramp.)		
There are no anatomical changes. It results from a deranged action of the nerve centers presiding over the muscles involved in writing.	*Spastic form.* Clonic or tonic spasm when the pen is grasped. *Tremulous form.* Tremor when the muscles are used. *Paralytic form.* Weakness and fatigue of the muscles.	Sensory and vaso-motor disturbances associated either with spasm, paresis, paralysis or tremor of the muscles employed in writing.
5. PSEUDO-HYPERTROPHIC PARALYSIS. (Pseudo-Muscular Hypertrophy.)		
There is no lesion of the nervous system. It is a primary affection of the muscles characterized by an excess of fat and connective tissue between the muscular fibres, and degenerative atrophy of the latter.	A disease of childhood manifested by enlargement of the muscles of the calf, thigh, buttock or back, and muscular weakness without sensory disturbances. The prognosis is unfavorable.	Gait (page 3). Enlargement of the muscles with weakness of the latter, and absence of the reaction of degeneration.

DIAGNOSIS OF DISEASES OF THE SKIN.

ERYTHEMATOUS INFLAMMATIONS OF THE SKIN.

Site of Predilection.	*General Character of the Eruption.*	*Characteristic Signs.*
1. ERYTHEMA NODOSUM.		
The extremities particularly the lower.	Bright red nodes varying in size from a pea to an egg.	Eruption accompanied by febrile disturbances. Nodes change color like a bruise.
2. ERYTHEMA MULTIFORME.		
The extremities.	The cutaneous lesions change frequently, and may consist of macules, papules, vesicles, or bullæ.	The variety of the lesions.
3. URTICARIA. (Hives.)		
The wheals occur mostly on the face and trunk, although other parts of the body are not exempt.	An inflammatory affection attended by the eruption of pale red, evanescent wheals.	Suddenness and short duration of eruption associated with intense itching.

VESICULAR INFLAMMATIONS OF THE SKIN.

1. HERPES ZOSTER. (Shingles.)		
Follows the distribution of the nerves.	Groups of small vesicles on inflammatory bases accompanied with neuralgia.	Grouping of vesicles and their relation to the nerves.
2. MILIARIA. (Prickly Heat.)		
On covered parts (breast, abdomen, extremities, etc.).	An inflammatory disease of the sweat glands, attended by a discrete eruption of minute vesicles.	Lesions are discrete and attended with burning, itching and perspiration.
3. ECZEMA. (Tetter.)		
The skin of any part of the body, with predilection for the face, genitalia, hands and feet.	A non-contagious affection with multiform lesions (erythema, papules, vesicles, pustules, scales and crusts).	Multiform skin lesions, itching and discharge.

BULLOUS INFLAMMATION OF THE SKIN.

Site of Predilection.	*General Character of the Eruption.*	*Characteristic Signs.*
1. PEMPHIGUS.		
May attack the skin or mucous membranes in any part of the body.	Characterized by the eruption of successive crops of bullæ or blebs. Bullæ are elevations of the skin containing serous fluid, and varying in size from a pea to an egg or an apple.	*Acute pemphigus* is an infectious disease, occurring in the new born. *P. foliaceus* is a fatal disease, characterized by bursting of the bullæ and the formation of thick crusts.

PUSTULAR INFLAMMATIONS OF THE SKIN.

1. IMPETIGO.		
On the face and extremities.	Eruption of discrete pustules varying in size from a pea to a cherry, and commonly observed in children.	Discrete pustular eruption which dries in a few days, leaving thin yellowish crusts, which drop off and leave a normal surface.
2. IMPETIGO CONTAGIOSA.		
On the face and extremities.	An acute contagious disease, occurring in debilitated children. The pustules are flat or umbilicated.	The yellowish crusts when they drop off leave behind an excoriated surface.
3. ACNE.		
The face and back.	Inflammatory disease of the sebaceous glands, characterized by papules and pustules, and associated with comedones.	Chronic character of the affection and implication of the sebaceous glands.
4. ACNE ROSACEA.		
On the face, especially the nose, cheeks, chin, etc.	Acne lesions associated with dilatation of the blood-vessels and tissue hypertrophy.	Acne lesions, dilatation of the vessels and overgrowth of tissue.
5. ACNE MENTAGRA. (Simple Sycosis.)		
Beard, eye-brows, and, rarely, the hair follicles of the head.	A chronic inflammation of the hair follicles, leading to tubercles, pustules, crusts and diffused infiltration of the skin.	Inflammation is superficial, the hairs are not involved, and the *trichophyton* is absent.

PUSTULAR INFLAMMATIONS OF THE SKIN—Continued.

Site of Predilection.	*General Character of the Eruption.*	*Characteristic Signs.*
6. TINEA SYCOSIS. (Barber's Itch.)		
Bearded region.	Like the foregoing. The hairs become lustreless, brittle and loose. If involved hairs are allowed to remain for fifteen minutes in a solution of caustic potash (1 : 3), the spores and threads of the *tricophyton tonsurans* can be seen.	Inflammation is deep, the hairs are involved, and the tricophyton can be demonstrated.

SCALY INFLAMMATIONS OF THE SKIN.

1. PSORIASIS.		
Extensor surfaces of the knees and elbows, hair of the head and ears. The lesions may appear on any part of the body.	A chronic inflammation leading to the development of pearly scales which, when detached, leave behind a red or bleeding surface. Itching is slight or absent.	Predilection for the extensor surfaces, dry pearly scales, absence of itching, protracted course and tendency to relapse.
2. PITYRIASIS RUBRA.		
Usually diffused over the body.	A rare disease of chronic duration, attended by tension and contraction of the skin, fever and the development of red spots which scale.	Fever attends the development of the eruption. The disease leads to marasmus.

PAPULAR INFLAMMATIONS OF THE SKIN.

1. PRURIGO.		
The back and extensor surfaces of the extremities.	A chronic disease attended by an eruption of small, discrete, pale red papules and itching. Develops in early childhood, and persists through life.	The development in early childhood, character of the eruption and intense itching.

PAPULAR INFLAMMATIONS OF THE SKIN—Continued.

Site of Predilection.	*General Character of the Eruption.*	*Characteristic Signs.*
2. LICHEN RUBER.		
Breast, abdomen, genitalia and flexor surfaces of the extremities.	A chronic disease occurring in middle-aged males, characterized by the development of small red papules, itching and failure of health.	Itching, failure of the general health, and its occurrence in poorly nourished middle-aged individuals.
3. LICHEN SCROFULOSIS.		
Back, breast, lower part of abdomen, and later the flexor surfaces of the extremities.	A chronic disease, occurring in children of a strumous diathesis, attended by small pale red papules.	Absence of itching, scrofulosis, pale skin, and occurrence in children.

HYPERTROPHY OF THE SKIN.

1. ICHTHYOSIS. (Fish Skin Disease.)		
Extensor surface of the extremities. May involve the skin over the entire body, excepting the flexor surfaces of the joints, genitalia, palms and soles.	A chronic affection manifested by dryness, thickening of the epidermis and scaliness. It is first detected in early childhood, and is an incurable disease.	Absence of inflammatory symptoms, chronicity and detection in early childhood.
2. SCLERODERMA.		
Upper half of the body, although it may be diffused.	A chronic affection leading to induration of the skin in patches, or involving the entire body. Death occurs usually from marasmus.	Induration of the skin, which renders the parts immobile.
3. ELEPHANTIASIS.		
Legs and genitals.	Hypertrophy of the skin and subcutaneous tissues, leading to enormous enlargement.	Thickening of the skin and subcutaneous tissues, associated with lymphangitis.
4. CHELOID. (Keloid.)		
Chest and back.	Hypertrophy of the skin, beginning as a pale red nodule, which sends out claw-like processes, making it resemble a crab. It returns after removal.	Occurs frequently in the colored race, history of heredity. It is distinguished from a scar by the fact that the latter does not extend beyond the injury.

ATROPHY OF THE PIGMENT OF THE SKIN.

Site of Predilection.	*General Character of the Eruption.*	*Characteristic Signs.*
1. VITILIGO. (Leucoderma.)		
On parts of the body exposed to injuries and pressure.	An acquired affection characterized by milk-white patches surrounded by areas of increased pigmentation.	Aside from the absence of pigment, the skin is normal.

NEUROSIS OF THE SKIN.

1. PRURITUS.		
Any part of the body.	A functional affection independent of any anatomical changes in the skin.	Itching and the absence of any definite skin lesions, unless they are the result of scratching.

PARASITES OF THE SKIN.

1. SCABIES. (Itch.)		
Between the fingers, wrists, axillæ, genitals, and beneath the mammæ. The face and scalp are never implicated.	A contagious disease caused by the *Acarus Scabiei*, and manifested by the development of papules, vesicles, pustules and burrows.	The itching is intense, history and sites of predilection. For demonstration of the *acarus*, see p. 189.
2. TINEA TRICOPHYTINA. (Ringworm.)		
Ringworm occurring on the *scalp* is known as *Tinea Tonsurans;* on the *body*, *Tinea Circinata:* on the *beard*, *Tinea Sycosis.*	*Tinea Tonsurans:* characterized by scaly, elevated patches, through which dry, brittle hairs project. *Tinea Circinata:* scaly patches which clear up in the center.	Itching and demonstration of the *tricophyton*, see p. 190.
3. TINEA VERSICOLOR. (Pityriasis Versicolor.)		
Chest and back. It may occur on the face, though rarely.	Salmon colored, yellowish or dark-brown spots, generally scaly, caused by the *microsporon furfur.*	Scaly pigmented spots and demonstration of the microsporon, see p. 190.

PARASITES OF THE SKIN—Continued.

Site of Predilection.	*General Character of the Eruption.*	*Characteristic Signs.*
4. PHTHEIRIASIS. (Pediculosis.)		
Pediculus capitis; lice confined to the scalp. *Pediculus corporis;* body or clothes lice. *Pediculus pubis;* crab louse confined to pubes, axillæ, etc.	Eczematous lesions and scratch marks caused by the intense itching; petechiæ caused by the bite of the insect.	Demonstration of the ova or nits and the lice. See page 189.
5. TINEA FAVOSA. (Favus.)		
The scalp. It rarely affects the nail substance (*onychomycosis favosa*).	A contagious scalp affection manifested by yellowish, cup-shaped crusts, through which dry, brittle, lustreless hairs project. The crusts have a peculiar musty odor.	Yellow, cup-shaped crusts, odor and demonstration of the fungus, *Achorion Schoenleinii,* see page 190.

DRUG EXANTHEMATA.

Quinine, morphine, opium, atropine, digitalis, chloral hydrate, antipyrine, phenacetine, sulphonal and *strychnine,* often produce an eruption of an *erythematous character.*

After the use of the *salicylates* a *bullous eruption* has been observed.

Arsenic may produce an itching *papular* or *vesicular eruption.*

Copaiba and *Turpentine,* produce an *urticaria*-like eruption.

Iodides and *bromides* produce an *acne* eruption.

BACTERIOLOGICAL DIAGNOSIS.

PATHOGENIC MICROBES.

Form and Arrangement.	*Growth.*	*Reaction.*
1. ACTINOMYCOSIS. (Ray Fungus.)		
Found in man and cattle as a round, flattened, hollow body, with a central cavity made up of club-shaped elements in the form of a rosette. In the pus or scrapings, little yellow grains about the size of a pin's head are seen containing the ray fungus.	Cultures obtained after five to six days on agar-agar and gelatine without liquefaction.	Cover glass preparations are best stained by Gram's method. (See page 193.)
2. ANTHRAX BACILLUS. (Bacillus Anthracis.)		
Found in the blood and organs of animals afflicted with splenic fever. The bacilli are rods with broad cup-shaped ends. In cultures long threads are formed with large oval spores.	Grows rapidly between 12° and 45° C., and requires plenty of oxygen. Colonies develop in two days on gelatine plates.	They take the aniline dyes. To bring out the cup-shaped ends, methylene blue is best.
3. CHOLERA BACILLUS. (Comma Bacillus.)		
Found in the intestinal canal and fæces of cholera patients. It is a curved bacillus about half the size of a tubercle bacillus. Discovered by Koch in 1884.	Develops at ordinary temperatures on all nutrient media that have an alkaline or neutral reaction. In a solution of peptone at 37° C., they develop rapidly on the surface in six to twelve hours.	It is best colored with aqueous solution of fuchsin, and may be decolorized by Gram's method. For color reaction, see page 123.
4. DIPHTHERIA BACILLUS. (Bacillus Diphtheriæ.)		
Found in diphtheritic membranes. Straight or crooked rods, the same length as the tubercle bacilli, but twice as thick. They stain most intense at the ends.	They grow on peptonized meat-sugar-serum infusion at 37° C. They grow readily on all media between 20° C. and 40° C.	After Löffler's method: few minutes in a concentrated alcoholic solution of methylene blue, to which has been added a KHO solution (1 to 10,000), then in one-half per cent acetic acid, then absolute alcohol, mount in balsam.

PATHOGENIC MICROBES—Continued.

Form and Arrangement.	*Growth.*	*Reaction.*
5. GONORRHEA. (Gonococcus.)		
Found in the secretions of gonorrhoic inflammation as cocci, always in pairs (*diplococcus*), biscuit-shaped, with flat surfaces lying together resembling the coffee-bean.	Growth doubtful. Only upon human blood serum have they given any semblance of growth.	Best stained with fuchsin or methyl-violet. It is not discolored by Gram's method.
6. LEPROSY. (Lepra Bacillus.)		
Found in all leprous products. Small rods, shorter than tubercle bacilli, with occasionally pointed ends.	Grows on blood serum to which peptone and glycerine have been added. Growth slow and requires about eight days at a temperature of 37° C.	Same reaction as tubercle bacillus (see page 65), but stain more quickly. They do not stain in Löffler's alkaline methyl blue solution, thus differing from the tubercle bacillus.
7. BACILLUS MALLEI. (Glanders.)		
Found in the nodules of the disease. Small rods about the size of the tubercle bacillus. The ends are rounded. They appear singly as a rule.	Grow abundantly on glycerine-agar or blood serum at 37° C.	Gram's method is not applicable. Löffler's methylene blue is the best stain.
8. MALARIA. (Plasmodium Malariæ.)		
Found in the blood corpuscles of malarial patients. They are protoplasmic corpuscles of various forms (round, oval, crescentic and flagella). They may or may not contain black pigment. They show rapid amœboid movements.	They have not been cultivated. Inoculations with the blood of a malarial patient will produce a typical attack of malaria in another person. They disappear from the blood after the use of quinine.	The organisms are best observed when the blood is fresh and unstained. A powerful lens is necessary. Dried specimens of blood may be stained with methylene blue.
9. PNEUMONIA. (Bacillus Pneumoniæ.) (Diplococcus Pneumoniæ.)		
Both forms have been found in pneumonia. The *bacilli* are oval-shaped rods nearly as wide as they are long. In tissues each bacillus has a faint capsule. The *diplococci* are rod-shaped, longer than broad, usually in pairs, and surrounded by a capsule.	The bacilli grow rapidly on all media. The diplococci grow slowly on alkaline media.	Stained by Friedlander's method (page 67), although the ordinary aniline stains will suffice.

PATHOGENIC MICROBES—Continued.

Form and Arrangement.	*Growth.*	*Reaction.*
10. RELAPSING FEVER. (Spirillum or Spirochæte Obermäieri.)		
Found in the blood of relapsing or recurrent fever. The organisms are long spiral threads with a distinct flagellum, and they move by means of wavy undulations. They are only found in the blood during the fever.	They have not been cultivated.	Ordinary aniline stains. After fixing in the flame, cleanse with a four per cent acetic acid solution and stain with the ordinary aniline solutions.
11. SUPPURATOIN. (Streptococcus Pyogenes.) (Staphylococcus Pyogenes Aureus.)		
Found in pus. They produce suppuration. The streptococci are cocci found singly and in chain-like groups. The staphylococci are micrococci in clusters like bunched grapes.	The streptococci grow in bouillon and the staphylococci on gelatine.	Ordinary stains and Gram's method.
12. STREPTOCOCCUS ERYSIPELATIS.		
Found in the lymph channels of the skin in erysipelas as very small cocci in pairs or chains.	Grows on gelatine, agar-agar and blood serum at a temperature from 30° C. to 37° C.	Retains color after Gram's method.
13. SYPHILIS. (Bacillus Syphilis.)		
A bacillus found in the tissues and secretions of syphilis. In size and shape it resembles the tubercle bacillus. It is frequently curved, and has clubbed ends.	Has not been cultivated.	Stain in fuchsin solution for twenty-four hours, rinse in water, then in a chloride of iron solution, then alcohol, and clear in clove oil.
14. TETANUS. (Tetanus Bacillus.)		
Found in the earth and in the secretions from the wounds of tetanus patients. They are fine bacilli, and form threads in irregular masses.	Develops slowly and only when oxygen is excluded. Grows in nearly all the media.	Colors with all the ordinary aniline dyes, and Gram's method.

PATHOGENIC MICROBES—Continued.

Form and Arrangement.	*Growth.*	*Reaction.*
15. TUBERCULOSIS. (Bacillus Tuberculosis.)		
Found in the products of human and animal tuberculosis as thin rods, one-fourth to one-half the diameter of a red blood corpuscle in length, beaded and with rounded ends.	Grows in glycerine agar-agar, on potatoes and blood serum. They grow slowly, and are only visible after ten to fifteen days.	See page 65.
16. TYPHOID FEVER. (Bacillus Typhosus.)		
Found in the blood, urine, fæces and tissues of typhoid patients as bacilli, three times as long as they are broad, with rounded ends.	They grow slowly on all nutrient media.	They take the aniline dyes less deeply than other bacilli. Not colored by Gram's method.

BACTERIOLOGICAL ANALECTS.

1. The diagnostic value of the *typhoid bacilli* when found in the stools is questionable, as it is almost impossible to differentiate them from the *bacterium coli commune* which is also present. Puncture of the spleen for the purpose of obtaining cultures of the typhoid bacilli is a procedure not always free from danger.

2. *Typhoid bacilli* retain their vitality for weeks in water. They thrive in the soil and are not killed by freezing.

3. The *spirilla of relapsing fever* are met with in the blood only during the paroxysms of fever, and disappear in the intervals. Their number is not proportionate to the severity of the attack. The spirilla are absent in the secretions.

4. The *streptococci of erysipelas* are found in the peripheral parts of the erysipelatoid inflammation, and almost exclusively in the lymphatic vessels.

5. The incubatory period of *inoculated erysipelas* is from fifteen to sixty hours.

6. The *bacilli of cholera Asiatica* occurs only in the stools, and never in the blood, of cholera patients.

7. The *bacilli of cholera nostras*, which resemble the bacilli of Asiatic cholera, are distinguished from the latter by being plumper and larger.

8. The *bacillus diphtheriæ* lies in the surface of diphtheritic pseudo-membranes, and is never found in the blood or viscera. Cover glass specimens may be made from the membrane.

9. The *plasmodium malariæ* may be found in the blood in the following forms: 1. An unpigmented hyaline body within the red blood corpuscles, which shows active movements. 2. A pigmented amœboid body inside the red corpuscle. 3. A segmenting body inside the red corpuscle. 4. Crescentic bodies within the red corpuscles. 5. Flagellate organisms.

10. The anæmia of malaria is caused by a destruction of the red blood corpuscles by the plasmodia.

11. In *empyema* an examination of the aspirated fluid is of great importance. If the *pneumococcus* is present, the case will progress favorably; if the *pus microbes*, staphyococci and streptococci are present, the empyema is of septic origin and the prognosis is exceedingly bad. If the fluid contains no *organisms*, it is, as a rule, of tuberculous origin.

12. The *diplococcus pneumoniæ* of Fraenkel is the most constant organism in croupous pneumonia, although found

in the buccal secretion in twenty per cent. of healthy individuals.

13. In the *secondary affections of pneumonia,* such as pleurisy, meningitis, etc., the *diplococcus pneumoniæ* is almost constantly present.

14. It is difficult to distinguish the *bacillus of syphilis* from the *smegma bacillus,* which is found beneath the prepuce.

15. The *bacillus leprœ* has been cultivated successfully, but inoculation experiments on animals have been unsuccessful.

16. The *anthrax bacilli* die rapidly under unfavorable conditions, while the spores possess a high power of resistance.

17. *Anthrax* in the human being occurs by inoculation through the skin in which case the disease is usually local (*malignant pustule*); through the lungs as in the wool *sorter's disease* (*pulmonic anthrax*), and finally through the intestinal canal by the reception of the virus mixed with food or water.

18. *Malignant œdema* is caused by a bacillus. It occurs in connection with compound fractures, deep wounds, and after hypodermatic injections. It is manifested by emphysema of the skin, putrefaction and œdematous softening of the muscles adjacent to the injury, and finally death.

19. The *tubercle bacillus* was discovered by Koch on the 24th of March, 1882. It is an anaërobic (living without air) bacterium. The transmission of tuberculosis is chiefly through the lungs.

20. If in doubt regarding the *diagnosis of glanders,* inoculation can be conducted on mice or guinea pigs. Ulcers form at the point of inoculation, followed by nodules, which caseate.

DISEASES OF THE INTESTINES AND PERITONEUM.

Etiology.	*Pathology.*	*Symptoms.*
1. APPENDICITIS.		
Occurs in young persons. More than fifty per cent. of the cases occur before the twentieth year. Fæcal masses and foreign bodies in the appendix play the most important etiological role. Traumatism is also a factor.	Inflammation of the appendix may be catarrhal or ulcerative with perforation. Fæcal concretions or enteroliths may be present in the appendix. Adhesions, peritonitis and abscess are sequences.	Sudden pain in the right iliac fossa, attended with fever, and local tenderness with or without fever. McBurney's point (page 118). If general peritonitis occurs, it is recognized by sudden abdominal pain, tympanites, increased fever and rapidity of the pulse.
2. CHOLERA INFANTUM.		
Prevails in hot weather and in artificially fed children. Faulty feeding and bad hygiene are predisposing factors.	An infantile disease of bacteritic origin (no specific microbe has been found), with inflammation of the mucosa of the stomach and intestines.	Vomiting, frequent watery discharge, high temperature (105° to 108° F.), rapid pulse and collapse symptoms.
3. CHOLERA MORBUS. (Cholera Nostras.)		
An acute sporadic disease. Changes in temperature, summer season and unripe fruit are the essential etiological factors.	A ptomaine or a specific bacillus may be responsible for the gastro-enteric catarrh.	A sporadic affection, characterized by vomiting, bilious purging, moderate fever and great prostration.
4. CONSTIPATION.		
Sedentary habits, atony of the intestines from local or constitutional causes, weakness of the abdominal muscles, etc.	The chief local cause is atony of the sigmoid flexure which forces the fæces into the rectum.	No special symptoms. Many symptoms of an indefinite nature have been noted as a result of the resorption of noxious matter (*copræmia*) and chlorosis is attributed to fæcal poisoning.
5. DIARRHŒA.		
Changes of temperature, ptomaines and irritating food in the intestines, bacteria, poisons, reflex disturbances, etc.	Catarrh of the intestines.	Frequent passages. In *membranous enteritis*, there are false membranes or mucous casts in the stools.

DISEASES OF INTESTINES AND PERITONEUM—Continued.

Etiology.	*Pathology.*	*Symptoms.*
6. DILATATION OF THE COLON.		
A congenital condition, or it may result from over-distension from fæcal accumulations.	Dilatation of the sigmoid flexure occurs, when it is congenitally long. An acute dilatation may follow a twist in the mesocolon.	Tympanitic distension of the abdomen. If the walls of the colon are hypertrophied, the colon may be felt. If distension of the colon is pronounced, it may press on the diaphragm and interfere with the heart.
7. DYSENTERY. (Bloody Flux.)		
Bad hygiene, cachectic conditions, exposure to cold, irritating food, etc. In the tropics, as well as in cold climates, the disease is produced by the *amœba coli*. See page 184.	The inflammation of the colon may be *catarrhal*, *diphtheritic* or *amœbic*. In connection with the latter form, abscess of the liver is a frequent sequela.	The passages of frequent blood-streaked stools are accompanied by tenesmus. In the amœbic or tropical form, the diagnosis is made by an examination of the stools.
8. ENTERO-COLITIS.		
Bad food and hygiene, and climatic conditions are predisposing elements.	Inflammation of the mucous membrane of the ileum and colon. The solitary and agminated glands are involved and often ulcerated.	Diarrhœa with blood and mucus in the stools, moderate fever, pain along the colon, and wasting of the body.
9. ENTEROPTOSIS.		
Dilatation of the stomach and mobility of the kidney are frequently associated with this condition.	Prolapse of the abdominal viscera, especially the transverse colon. When all the viscera fall down the condition is called *splanchnoptosis.*	Constipation from narrowing of the colon, and motor, sensory and vascular disturbances from falling down of the stomach and intestines.
10. INTESTINAL COLIC. (Enteralgia.)		
As a *symptom* it occurs in plumbism, intestinal obstruction and inflammation. It may be produced by flatulence, fæcal accumulation or irritating food.	A functional affection caused by spasmodic contraction of the intestines.	Severe spasmodic pain localized around the umbilicus, relieved by pressure and the discharge of wind. Unattended by fever as a rule.
11. INTESTINAL ULCERS.		
Ulcers may be specific from syphilis, tuberculosis or typhoid fever. Ulceration may also arise from the various forms of intestinal inflammation and pressure of fæces (*stercoral ulcers*).	The duodenal ulcer is of peptic origin, like the round ulcer of stomach. The site of tuberculous and typhoid ulcers is the ileum, while the ulcers of dysentery are located in the large intestine.	The diagnosis is often difficult, and the case may furnish no symptoms. The following symptoms are of note: diarrhœa, pain, and blood and pus in the stools.

DISEASES OF INTESTINES AND PERITONEUM—Continued.

Etiology.	*Pathology.*	*Symptoms.*
12. INTESTINAL OBSTRUCTION. (Ileus.)		
Acute obstruction may be caused by: 1. Congenital occlusion. 2. Strangulation. 3. Twists (*volvulus*). 4. Intussusception. *Chronic obstruction*: 1. Fæcal accumulations. 2. Strictures. 3. Foreign bodies (gall-stones). 4. Tumors.	*Strangulation* is the most frequent cause of acute obstructions, and the site is usually in the small intestines. Twists are associated with a very long mesentery. The most usual site of intussusception is at the ileo-cæcal valve, as it descends into the colon.	*Symptoms of acute obstruction*: constipation, pain in the abdomen and vomiting. The degree of abdominal distension depends on the site of obstruction. *Chronic obstruction*: same as foregoing, although the development of symptoms is slow.
13. PERITONITIS.		
Primary peritonitis may arise from exposure to cold or traumatism. It may be *secondary* from extension of inflammation from neighboring organs. *Chronic peritonitis* may be caused by tuberculosis, syphilis, cancer, etc.	According to the character of the exudation, the inflammation may be: 1, fibrinous; 2, serofibrinous; 3, purulent; 4, putrid; 5, hemorrhagic.	Moderate fever, abdominal tenderness, abdominal distension, vomiting, pinched features and constipation.
14. PSOROSPERMIASIS.		
Common in the invertebrates and rare in the higher mammals. The parasites are called *psorosperms*, and they produce in man a disease similar to that occurring in rabbits.	In *internal psorospermiasis*, nodules resembling tubercles and containing *psorosperms* are found upon the peritoneum, omentum, liver and other viscera.	Fever of an intermittent type, diarrhœa, tenderness of the affected organs, and usually death. In *cutaneous psorospermiasis*, there are papillomatus growths containing psorosperms.
15. TYPHLITIS STERCORALIS.		
Caused by the lodgment of the fæces and occurs especially in young persons who have been constipated or suffered some error in diet.	An inflammation of the cæcum. Many cases of typhlitis are probably due to disease of the appendix.	Pain in the right iliac fossa. A sausage-shaped doughy tumor is present in this fossa. The fever is moderate. Prognosis is favorable.

ANALECTIC REVIEW OF GASTRIC DIGESTION AND GASTRIC NEUROSES.

THE PROCESS OF DIGESTION.

1. The first period of digestion in the stomach is the *amylolytic*, and consists in the conversion of *starch* into *dextrine* (achroö- and erythro-dextrine). The digestion is effected in the stomach by the swallowed saliva.

2. *Grape sugar*, which is a final product of starch digestion, is converted into *lactic acid* by the action of microorganisms that excite fermentation.

3. The *amylolytic period of digestion* lasts, on an average, three-quarters of an hour.

4. *Muriatic acid* and *pepsin* are secreted immediately after the ingestion of food.

5. When *muriatic acid* is first secreted it is in combination, and the *free acid* is not present until three-quarters of an hour after the ingestion of food.

6. Free *muriatic acid inhibits* amylolytic digestion, as the digestion of starch cannot proceed in an acid medium.

7. *Lactic acid disappears* from the stomach one hour after the ingestion of food, or at a time when free muriatic acid is present.

8. Simultaneous with the free hydrochloric acid, the milk curdling ferment (*rennet ferment*), which coagulates the casein of milk, and pepsin, are secreted.

9. *Pepsin* and *rennet ferment* are not secreted as such, but are formed by their ferments, *propepsin* and *rennet zymogen* into pepsin and rennet ferment, respectively, under the influence of muriatic acid.

10. The second or *muriatic period of digestion* consists in a *peptonizing action* of the gastric secretion upon the albuminous bodies.

11. The *free muriatic acid* is *antiseptic*, destroying all microörganisms concerned in fermentation and putrefaction.

12. During the period of digestion, some of the digested food is absorbed, while the remaining portion slowly passes into the duodenum.

13. About six hours after a mixed meal, the stomach should be practically empty.

14. In the *intervals of digestion*, the stomach contains only a small amount of clear fluid of neutral reaction.

EXAMINATION OF GASTRIC DIGESTION.

1. To determine the *duration* and other facts concerning digestion, the patient takes the experimental meal of Leube.

2. *Leube's experimental meal* consists of a plate of water-gruel, an ordinary piece of beef-steak, and white bread.

3. Seven hours after the meal of Leube, if digestion is normal, the stomach should only contain a few remnants of food.

4. After removal of the gastric contents, examinations must be made for the following: *free muriatic acid, lactic acid, butyric* and *acetic acids*, determination of *the total acidity*, determination of the *digestive power of the gastric juice*, its *coagulating effect* upon milk, the *transformation* of *starch*, the presence of *peptone* and *propeptone*, the *absorptive* and *motive power* of the stomach.

5. The most certain test for *hydrochloric acid* is the *phloroglucin-vanillin test*. It consists of phloroglucin two parts, vanillin one part, to thirty parts of absolute alcohol. One or two drops of this solution heated on a porcelain plate with the gastric contents (filtered), will, if free hydrochloric acid is present, give a red color, the intensity depending on the percentage of the acid. It is distinct to 0.05 per thousand.

6. The *phloroglucin-vanillin test*, or *Günzberg's reaction*, is only interfered with if sulphuretted hydrogen is present.

7. *Lactic acid* is detected by the solution of *Uffelman* (to about three ounces of a two per cent. solution of carbolic acid, add one or two drops of chloride of iron solution). This steel-blue mixture becomes yellow or yellowish-green if lactic acid is present. It becomes like water if only HCL is present. The test may be counterfeited in the presence of phosphates, mineral acids, alcohol and grape sugar. These errors may be removed if the gastric secretion is shaken with ether. After evaporating the ether, dissolve the ether residue in water and apply test.

8. *Butyric* and *acetic acids* are recognized by their odor. They convert *Uffelman's* solution into a yellowish-red color.

9. The *total acidity* of the gastric contents is determined by neutralizing it with a one-tenth normal solution of soda. 1 c.c. of the soda solution neutralizes 0.00365 of hydrochloric acid.

10. The *digestive power* of the gastric juice is determined by the digestion of a piece of the white of a hard-boiled egg or a bit of washed fibrin. See page 113.

11. The *coagulating effect* of the gastric juice due to the *rennet ferment* is demonstrated as follows: add three to five drops of the filtered gastric juice to 10 c.c. of milk, when curdling will occur, in about fifteen minutes, at 37° to 40° C.

12. The *transformation of starch* is tested by adding a drop of *Lugol's solution* (iodine one part, iodide of potash two parts, aq. dest. two hundred parts) to the filtered gastric juice. If *starch* is present the solution becomes blue, if *erythrodextrin*, purple. A mixture of starch and dextrin with the first few drops of the solution becomes colorless, but upon adding more of the solution, it becomes red and then blue.

13. The test for peptone is the *biuret reaction.* See page 112.

14. Testing the *absorptive* and *motive* power of the stomach is described on page 113.

ANOMALIES OF GASTRIC DIGESTION.

1. *Anacidity* or *inacidity* denotes a condition in which free hydrochloric acid is absent. It occurs in destruction, atrophy and degeneration of the mucous membrane of the stomach, in gastric cancer with dilatation, in severe anæmia, in febrile conditions, and in certain cases of nervous dyspepsia.

2. Anacidity interferes with the digestion of albumen and the absence of hydrochloric acid favors putrefaction and fermentation.

3. *Hyperacidity* or *superacidity* is a condition in which the free hydrochloric acid is present earlier than usual and is in excess. It occurs in certain nervous dyspepsias (*pyrosis hydrochlorica*), and in acute and chronic gastric catarrh.

4. Hyperacidity interferes with the digestion of starch. Albuminous bodies are usually rapidly peptonized.

5. *Chronic hypersecretion* signifies a continuous secretion of gastric juice even though the stomach is empty. The fluid present resembles gastric juice with a high degree of acidity.

GASTRIC NEUROSES.

1. When a patient has a ravenous appetite which he finds difficult to satiate, the condition is called *bulimia* or *polyphagia*. It may occur alone or as a symptom of many nervous affections, neurasthenia, hysteria, psychoses, etc. It is likewise a prominent symptom in *diabetes*.

2. *Pica* is a condition of perverted appetite often symptomatic of *hysteria*, in which the patient craves for ink, chalk and other substances.

3. *Anorexia* is a lack of appetite or repugnance for food. It accompanies many dyspeptic and mental disturbances.

4. *Gastralgia* or *Gastrodynia* is a neuralgic affection of the stomach dependent on irritation of the sensory nerves. It may be symptomatic of *tabes* (gastric crises), hysteria, neurasthenia, primary anæmia and various mental diseases.

5. An increased secretion of hydrochloric acid (*hyperacidity*) may exist as a primary neurosis of the stomach.

6. *Nervous belching* occurs in hysteria and neurasthenia.

7. *Pneumatosis* is a condition in which the stomach is inflated with air often conducing to typical asthmatic attacks (*asthma dyspepticum*).

8. *Nervous vomiting* is frequent in hysteria. It is not attended by severe nausea or retching.

9. *Peristaltic unrest of the stomach* is a condition usually dependent on *gastrectasis*, in which the peristalsis of the stomach is so marked, that the movements may be observed through the relaxed abdominal parietes. The movements may be attended by loud rumbling sounds.

10. *Nervous anacidity* is often encountered as a pure neurosis in hysteria and neurasthenia.

11. *Incontinence* of the *pylorus* and *cardia* are conditions likened to paralysis. Paresis of the latter allows of the regurgitation of air and food, while paresis of the former allows the undigested food to pass at once into the intestines.

12. *Incontinentia pylori* may be recognized by distending the stomach with air or gas (tartaric acid and bicarbonate of soda). It will be noted that the stomach becomes incompletely distended, and that the air or gas passes at once into the intestines.

13. *Rumination* or *merycismus* is a neurosis in which food, after it is ingested, returns to the mouth to be chewed and swallowed a second time.

14. *Atony of the stomach* may be partial or complete. It is suppressed peristalsis of the stomach.

RELATION OF DISEASES OF THE STOMACH TO OTHER DISEASES.

Anæmia.—Absorption and peristalsis of the stomach are disturbed, leading to a complex variety of digestive disturbances.

Cardiac Diseases.—As a result of venous congestion, catarrh of the stomach occurs.

Diabetes.—Dyspeptic disturbances frequently serve, as an index to this disease. Hydrochloric acid, when temporarily absent, may be merely an expression of a neurosis; if absent permanently, the cause is atrophy of the mucous membrane, the result of interstitial inflammation.

Gout.—There may be a specific gouty disorder of the stomach, the result of the uric acid diathesis, or the stomach may participate in the symptoms of this affection.

Hepatic Diseases.—Many affections of the liver may be disguised as a chronic gastric catarrh. There is an intimate relationship existing between the functions of both organs.

Malaria.—Aside from dyspeptic disturbances as manifestations of this disease, there are typical neuroses with characteristic periodicity, which yield to quinine.

Nervous Diseases.—In tabes dorsalis, paroxysms of gastralgia and gastric crises occur not only in the late, but in the early stages of the disease. Gastric crises are severe paroxysms of pain, confined to the epigastrium, accompanied by painful and uninterrupted vomiting, nausea and vertigo. A characteristic feature of the crises is this, that almost immediately after the pains cease, the patients demand food, which they digest perfectly.

Renal Diseases.—Disturbances of digestion and vomiting occur as a result of the excretion of excrementitious products by the stomach, symptoms in reality of uræmia. Mobility of the kidney may cause gastrectasis by compression of the duodenum.

Tuberculosis.—Many cases of phthisis begin with dyspeptic disturbances; in fact, Hutchinson maintains that dyspeptic symptoms precede the onset of tubercular manifestations in thirty-three per cent. of the cases. Fenwick found evidences of gastric catarrh in seventy-three per cent. of his phthisical cases.

THE EMPLOYMENT OF DRUGS IN DIAGNOSIS.

Cocaine.—As a local anæsthetic, this drug subserves a useful purpose in diagnosis. Recurring or persistent headaches, as well as a large group of neuroses, are often dependent on some abnormity in refraction, or to an improper balance between the sets of muscles which regulate the ocular movements. A solution of cocaine, sprayed on the ocular conjunctiva, paralyzes the accommodation, and will, in many instances, for the time being, ameliorate or cause to disappear the reflex symptoms dependent on the ocular defect. For examining the back ground of the eye, dilatation of the pupil had better be secured by cocaine than by atropine, inasmuch as the latter paralyzes the accommodation for too long a period. Any irregularity in the dilatation of the pupil may direct attention to an old iritis. Among the *reflex nasal neuroses,* the following may be cited: headache, vertigo, insomnia, neuralgic affections in typical situations (scapula, sternum, renal region), and disturbances in the visceral functions (asthma, palpitations, vomiting, etc.). If the nasal mucous membrane is anæsthetized by cocaine, many of these neuroses will disappear, thus furnishing an invaluable aid in diagnosis. Various reflex troubles may originate from *urethral irritation,* especially from stricture. These reflex troubles may merely amount to pains in the perineum, abdomen, and in other remote parts, or they may constitute such affections as neurasthenia, epileptiform convulsions, etc. The urethral

instillation of a solution of cocaine may prove an aid in diagnosis. Similarly, reflex rectal, uterine, vaginal and laryngeal troubles may be determined.

Nitrite of Amyl.—The physiological action of this remedy is to produce vascular dilatation. It is administered by inhalation. The pains of *angina pectoris* are frequently relieved by the inhalation of this substance, and it may serve as a means of differentiation from *pseudo-angina*. *Headaches* of anæmic origin are relieved, whereas, if the headache is of hyperæmic origin, it is intensified. *Spastic migraine* is relieved, while paralytic migraine is intensified. *Neuralgias* of anæmic origin are likewise benefited. I have used inhalations of amyl nitrite in differentiating the râles of *asthma* from those of the accompanying *bronchitis;* the former disappear, while the latter persist. In many pulmonary neuroses of spasmodic character, nitrite of amyl inhalations are of signal advantage in aiding the results of auscultation.

Iodide of Potash.—This remedy, like mercury, is almost diagnostic when benefit is secured from its administration in neuralgiæ, ulcerations, paralyses, etc., of *syphilitic origin*. It must be given alone, and in large doses, until its physiological action is secured. It is also used in the diagnosis of suspected *pulmonary tuberculosis* when in apical lung affections, dullness or modified respiration is present without râles. The latter may be artificially produced by its administration. When the cough is dry without sputa, the latter may be produced by giving iodide of potash. To intensify the auscultatory phenomena of an old *pleuritis*, iodide of potash, by increasing the secretions, may be of some value. It is also used for determining the absorptive power of the stomach (see page 113). The value of potassium iodide in the removal by resorption of hydatid cysts is determined by its administration in a given case. If aspiration of the sac shows the presence of iodine, then the potash treatment will be of value, otherwise, the results will prove negative.

Iron.—The ferruginous preparations are of diagnostic value in determining the *anæmic origin* of neuralgiæ, headaches, cardiac murmurs, etc. They should disappear or become ameliorated after their persistent use. In *chlorosis,*

iron is a specific. Iron intensifies the symptoms of any affection due to *plethora*. It is always better to look upon anæmia, not as a substantive, but as a symptomatic affection. I have shown elswhere that *pulmonary atelectasis* (page 50) is a frequent factor in the etiology of anæmia, and furthermore, that by persistent, daily, methodical lung gymnastics, many intractable forms of anæmia may be cured.

Digitalis.—This drug, by slowing the action of the heart, is of value in irregular action of that organ, to determine the time of a *murmur*, should this be present. At the same time, by increasing the force of the heart's action, it will render *cardiac murmurs* more distinct, inasmuch as the intensity of a murmur is dependent on the activity of the heart. *Fluid in the pericardium* may be an *exudation* or *transudation*. Digitalis, by increasing diuresis, will often cause the resorption of the latter, leaving the former unaffected. Resorption is noted by a diminution in the area of præcordial dullness. In *tachycardia*, resulting from vagus paralysis, the heart does not respond to digitalis.

Quinine.—In the typical forms of *malaria*, the diagnosis is always easy; but there are types of the disease in which the febrile manifestations are irregular. In the latter cases, the therapeutic test of quinine is unquestioned, for the symptoms of malaria, however unusual, always yield to quinine. Any intermittent fever which does not respond to quinine is not malarial. In applying this therapeutic test, we must be lavish with our drug, always carrying it to its physiological effects, and maintaining them for several days.

Colchicum.—This is a specific palliative in *acute gout*. There are cases of irregular gout dependent on a so-called gouty diathesis, with a train of manifestations implicating nearly every organ of the body. Among the symptoms, headache and neuralgia are frequent. It is in the irregular cases of gout, that the colchicum test should be applied, coupled with the other routine methods of treatment.

Ergot.—The action of this drug is to produce arterial anæmia, and its use is suggested in the differential diagnosis of anæmic and congestive affections. The latter will be ameliorated.

Salicylic Acid.—This preparation is almost a specific in rheumatic affections, especially in the acute cases. In atypical manifestations of presumable rheumatic origin, the salicylates should always be given as a therapeutic test.

Salol.—This agent is used for determining the *motor activity of the stomach.* It is a compound of phenol and salicylic acid, and it is only split up in an alkaline medium. After taking fifteen grains of salol in a capsule, salicyluric acid will be detected in the urine in from forty to seventy-five minutes; which shows that the salol has passed out of the stomach. The acid may be easily detected by placing a drop of urine on filter paper; if to the latter a drop of a ten per cent. ferric chloride solution is added, a violet color appears. In atony of the stomach and gastric dilatation, the salicyluric acid may not be detected in the urine for many hours. There are many cases of *diarrhœa* due to bacteritic intestinal infection. A diarrhœa of this kind responds favorably to salol or any other intestinal antiseptic.

Opium.—This drug will relieve *dyspnœa* produced by many painful thoracic affections.

Anæsthetics.—They are employed in the detection of simulation of various kinds. It may only be necessary to carry the action of the anæsthetic up to the stage of excitement.

Hysteria is an affection which may simulate almost any disease, and the detection of the malady is often possible only after narcosis. Hysterical contractures and joint affections are examples.

A *phantom tumor* is of hysterical origin. It is produced by relaxation of the recti muscles and spasmodic contraction of the diaphragm in conjunction with gaseous inflation of the intestines, and an arching forward of the vertebral column. This tumor always disappears under full anæsthesia. A thorough palpation of the abdomen is only possible, in many cases, after complete relaxation of the abdominal parietes under an anæsthetic.

Blisters.—They are of value in diagnosis when it is necessary to secure the blood serum for examination, as in the application of Garrod's test (page 100). The serum of

blisters produced over suspected *leprous patches* may contain the bacilli of leprosy.

Anthelmintics.—There are many nervous phenomena, such as epilepsy, convulsions and chorea, which may be caused by intestinal parasites. In all cases of doubt regarding the etiology of a neurosis, intestinal parasites should be excluded by the administration of an anthelmintic.

Parasiticides.—There are many skin affections of a parasitic origin, the nature of which can always be satisfactorily determined by the application of parasiticides.

Jaborandi.—This powerful diaphoretic is usually employed in the form of its alkaloid, *pilocarpine.* It is not infrequent, especially in children, that a *pleuritic effusion* may yield the same physical signs as a pneumonia, in which case a differentiation may be necessary without having recourse to the exploratory needle. If the upper line of dullness is marked and then an hypodermic injection of pilocarpine is given, the line of dullness on percussion will be lower after than before the injection. *Pulmonary œdema* may be differentiated from the first stage of pneumonia and capillary bronchitis in a similar manner, the râles of the former disease often disappear for a limited time, at least, after the injection.

RECENT METHODS OF DIAGNOSIS.

Intra-Thoracic Auscultation.—Dr. B. W. Richardson employs for this purpose an œsophageal tube with a large aperture at the end. An aperture in the tube is necessary, otherwise there will be poor conduction of sound. The free end of the tube is connected with a binaural stethoscope. This method, according to the writer, is found applicable in diagnosing pulsating tumors (*aneurisms*) as they pulsate along the œsophagus and stomach. It is also of service in diagnosing diseases of the heart. Recourse is not had to this method unless the conventional methods yield negative results.

A New Method of Auscultatory Percussion.—This method, advocated by Dr. A. H. Smith, may be con-

ducted with the usual binaural stethoscope, to which is attached the small extremity intended for examining the heart, but this, instead of being applied to the chest, is held between the patient's teeth, and the lips are closed around it. During the percussion the nostrils are compressed with the fingers. By this method there is a confined column of air, reaching uninterruptedly from the patient's lung to the examiner's ear. I have employed this method of examination and can recommend it, when it is desirable to intensify the sounds obtained by ordinary percussion. The pneumatoscope described on page 46, embodies the principle of the method just described, but it is more complicated and yields poorer results.

Determining the Lower Liver Border by Auscultation.—This method, described by C. Verstraelen, is based on the fact that the intensity of the heart tones is well preserved over the entire hepatic region; at the lower border of the liver, however, the tones are suddenly lost. In this way, the lower liver border is determined. The abdominal walls must be relaxed, otherwise they will conduct the heart tones. I consider this an imperfect method of examination, especially if the heart tones are weak. The demarcation of the lower liver border by percussion is very difficult, if not often impossible, in the epigastric region, owing to the relative thickness of the abdominal parietes and the thinness of the liver border. My method of determining the lower liver border by linear percussion is as follows: the patient is placed in a position favoring the approximation of the liver to the abdominal walls. This position is the knee-elbow one, or if this is impossible, by the erect posture, the body being inclined forward. The edge of a large coin is then deeply imbedded in the abdominal walls, and the free edge of the coin is percussed lightly until a dullness is obtained. If this dullness represents the liver border, then, in accordance with the fact that the liver undergoes respiratory dislocation, the line of dullness will be lower during inspiration and higher during the act of expiration. The essential principle embodied in my method is this, to verify the correctness of my percussion by dislocation of the organ during respiration. This method will be found applicable in the percussion of the anterior border of the spleen, remembering, however, the addi-

tional fact, that change of posture will also influence the position of this viscus.

A New Diagnostic Sign of Stenosis of the Œsophagus.—According to Dr. Vanni, a sub-tympanitic sonorous sound is discovered by percussion on the left side of the vertebral column, the area affected occupying a rectangular space with a longitudinal axis. This resonance is changed into dullness when the patient has drunk a large quantity of fluid. If the œsophageal sound is then introduced down to the point of the stricture, it brings out a muddy and fetid fluid. Besides these diagnostic signs, the following phenomenon is noted: on assuming a reclining position, the pulsations of the femoral artery diminish in volume and strength when the patient drinks a quantity of fluid exceeding thirteen ounces; and if this liquid is slowly withdrawn, by means of the stomach pump, the beats of the femoral artery also slowly resume their normal amplitude.

The Movements of the Diaphragm.—The movements of this muscle have, heretofore, been determined by percussion. According to Sutton, they may be noted in nearly every individual. They are recognized by undulatory movements, beginning on either side at the sixth intercostal space, descending a number of intercostal spaces during deep inspiration, to rise again during expiration. The play of the diaphragm can also be observed in the back. For the proper observance of these movements on the anterior surface of the chest, the patient should be in the recumbent position.

New Methods of Diagnosis in Aneurism.—If the extremities are tied, or if the femoral and axillary arteries are compressed, the pressure symptoms of an aortic aneurism become intensified. This is also true regarding the signs obtained by auscultation. *Tracheal tugging* is a valuable sign, first described by Porter. To test this, the patient is placed in the erect position and directed to close the mouth and elevate the chin to the fullest extent, then the cricoid cartilage is grasped between the thumb and finger and gently pressed upwards, when, if *dilatation* or *aneurism* exists, the pulsation of the aorta will be distinctly felt, transmitted through the trachea to the hand. Dr. Wm. Evart has recently studied the occurrence of this

symptom in healthy individuals. He found that, in health, the symptom is only slightly, if at all present. Drummond directs attention to the occurrence of a systolic murmur which may be heard in the trachea, or at the patient's mouth, when opened. It is either the sound conveyed from the sac, or is produced by the air as it is driven out of the trachea during the pulsations of the aneurism.

Osler describes another important symptom in large thoracic aneurisms, and that is, obliteration of the pulse in the abdominal aorta and its branches. In these cases, the sac probably acts as a reservoir, annihilating the ventricular systole and converting the intermittent into a continuous stream.

The Clinical Value of Vertebral Tenderness.—In *neurasthenia*, tenderness over the vertebræ is rarely absent. In *acute transverse myelitis*, there is frequently a zone of hyperæsthesia at the level of the lesion, which may be discovered by passing a test-tube containing hot water along the spine, when the sensation of warmth changes to one of actual pain. In *round ulcer of the stomach*, Boas claims that there is a painful dorsal point of great diagnostic value. It is found at the level of, and to the left of, the tenth to the twelfth dorsal vertebræ. The painful area lies usually directly against the vertebra. In no other disease of the stomach is such a painful area to be found with equal constancy. Another as yet undescribed painful area described by the same author, is found in *cholelithiasis*, in the back, at the region of the twelfth dorsal vertebra, and somewhat to the right side; this is found at the time of the attack, and even weeks and months thereafter. In *caries of the vertebræ*, the spinal processes of the affected vertebræ are painful on pressure, and pain is experienced when the vertebral column is moved. The muscles in the implicated district are nearly always rigid. In *intercostal neuralgia*, percussion of the vertebræ corresponding to the affected nerves will always elicit pain.

Ehrlich's Method of Examination of the Blood.—In staining blood specimens, the hæmoglobin must first be fixed on the cover glasses, but, instead of resorting to the complicated, although thorough method, as described on page 99, the cover glasses may be dropped into

a solution of absolute alcohol and ether of equal parts. In a half-hour the specimen is ready to stain. The most convenient stain for every day use is the following solution :

Saturated aqueous solution of orange—G	125
Saturated solution (in 20 per cent alcohol) of acid fuchsin	125
Absolute alcohol	75
Saturated aqueous solution of methyl green	125

The methyl green must be added drop by drop while stirring or shaking the solution. The solution must stand some weeks, and the fluid used should be taken with a pipette from the middle of the solution. The cover glasses are stained in this solution for from two to five minutes, washed in water, dried, and mounted in oil and balsam. With this stain, the red corpuscles are stained an orange or buff color, the nuclei of the colorless corpuscles green, the eosinophilic granules a deep red ; the nuclei of the nucleated red corpuscles, when present, deep green, almost black. The diagnostic value of the *eosinophile leucocytes* in leukæmia has lately been questioned. They have been found in gonorrheal pus, purulent ophthalmia, human pus, in nasal polypi, and finally in the sputum of patients suffering from asthma, phthisis and bronchitis.

The Hæmatokrit.—This instrument is so constructed as to separate by centrifugal force the blood corpuscles from the containing serum, the former being of greater specific gravity.

One turn of the large wheel of this instrument causes the brass frame at the top of an upright piece of steel to revolve one hundred and four times. The frame receives two glass tubes, which are to contain the blood, each measuring thirty-five millimetres in length, and held securely in position by a spring. The glass tubes have a capacity of about twenty-seven and a half cubic millimetres, are three and three-fourths millimetres thick, thirty-five millimetres long, and have a calibre throughout measuring one millimetre in diameter. A scale on the outside divides it into fifty equal parts, in the same manner as the scale on an ordinary thermometer.

To obtain the volume of corpuscles : the blood is mixed with an equal quantity of a fluid preventing coagulation, and is then rotated ; the red corpuscles form a column at

the periphery of the tube, and measure, let us say, twelve and a half. As the blood is diluted one-half, this result is multiplied by two, equal to twenty-five, and to convert this into percentage it is again multiplied by two, as the scale is divided into but fifty parts, which gives fifty per cent.

After considerable experimentation, Dr. J. Daland has concluded that a two and a half per cent. solution of potassium bichromate is by far the most useful liquid to dilute the blood for counting red blood-corpuscles. The method of applying the hæmatokrit is extremely simple, and requires very little additional apparatus.

The hæmatokrit gives more accurate results than the apparatus of Thoma-Zeiss (page 96), and requires less skill and time in its employment.

The Centrifugal Machine.—This apparatus is designed for the purpose of separating the corpuscular elements of fluids by centrifugal force. The motion is produced by two cog-wheels, that put in action a large beveled friction-wheel of rubber, causing a vertical spindle to revolve rapidly; to the latter a horizontal arm is connected, equally divided from a common centre, and containing two glass tubes, into which the fluid for separation is placed. By means of this apparatus casts can be separated from the urine in a few minutes. By means of this apparatus, I have frequently discovered elements in the urine, which, by the ordinary method of sedimentation by gravity, would never have been discovered. The *galvanic current* has also been used for obtaining a sediment. The current obtained from two zinc-carbon elements is passed, by means of platinum electrodes, through urine or other fluids contained in a bottle-shaped flask, during five to ten minutes. A layer of froth due to disengaged gas appears at the surface, and beneath this a turbid layer. If the current is allowed to act longer, the sediment sinks to the bottom.

Ptomaine Poisoning.—Ptomaines are poisonous compounds developed from bacterial decomposition of animal matters. They are frequently ingested with the following tainted foods:

1. *Shell-fish and fish.*—This follows the eating of either raw or cooked mussels, stale oysters, salted sturgeon, etc.

The symptoms are those of acute poisoning, with symptoms of collapse. The poison of the mussel is *mytilotoxin* which exists in the liver of the mussel.

2. *Milk.*—The substance which causes the poisonous symptoms is *tyrotoxicon.*

3. *Meat.*—Sausage poisoning (*botulism*) is relatively frequent. The symptoms are those of gastro-intestinal irritation, accompanied by headache, prostration, thirst, abdominal pain, excessive sweating and collapse.

Illumination of the Stomach.—Renvers recently demonstrated to the Berlin Medical Society an apparatus which consisted of a small Edison's lamp fixed to the end of a bougie and covered by a small glass case filled with water.

The electric current is obtained from a battery of twenty cells. If the stomach is full of food, illumination is impossible, but if it is quite full of water when the lamp is passed into the stomach an illuminated area is seen, which corresponds exactly to the limits of the organ. The apparatus can only be used in the erect posture, when the greater curvature is usually seen a little below the level of the umbilicus.

Abnormal dilatation of the stomach can be readily detected, and a case of carcinoma of the organ has been diagnosed by the use of this apparatus, the diagnosis being confirmed by *post mortem* examination. The tumor appeared as a dark spot in the light field.

Transillumination as a Means of Diagnosis.—Antral empyema has been diagnosed by illuminating the bones of the face by an incandescent lamp introduced into the patient's mouth. Darkness on one side of an illuminated face might be taken as a probable symptom of empyema. The thickness of the bone varies so greatly in different individuals that it cannot be considered a reliable test. The diagnosis of *pus in the antrum* is often made by the simple method of Fränkel. The nose having been thoroughly cleansed, and it having been ascertained by inspection that the nostril corresponding to the suspected antrum is quite free from secretion, the patient is instructed to lie down across a chair, holding the head down with the

affected side uppermost. After remaining in this position for half a minute, he is told to sit up, when an examination is made. Pus in the middle meatus will be noted if the discharge comes from the antrum. If a small incandescent lamp be inserted in the auditory meatus, in a dark room, a *healthy mastoid* is immediately illuminated with a ruddy glow. If the mastoid cells be occupied by a purulent collection, the glow will be absent and the cells dark. Comparison with the healthy side should always be made.

The Virile Reflex.—Dr. Hughes has recently directed attention to this reflex, which is obtained as follows: the penis is taken and pulled with some little force upwards in the direction of the umbilicus; the foreskin is then caught near the frænum, and the corpus cavernosum grasped with the second and third fingers; if the corpus cavernosum is now struck with the other hand, a jerk is elicited. When this reflex is not obtained, the patient is said to be addicted to masturbation, or there is some loss of sexual power.

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